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TREATMENT OF MALARIA.[1]

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TREATMENT of malaria is nowadays such a large subject that in a paper such as this, it is possible only to touch on broad outlines. The action of the various antimalarial remedies and their use in treatment of acute attacks and of recurrences will be dealt with; but, as *ovale* tertian and quartan malaria are so rare in the South Pacific zone, discussion will be confined to the benign tertian (*Plasmodium vivax*) and subtertian (*Plasmodium falciparum*) types.

From the standpoint of prevalence, malaria is the most important of all diseases in the world today. Records of the Health Organization of the League of Nations, with figures from sixty-five countries, showed that 17,750,000 patients suffering from malaria were treated in 1932.

In India, with a population of 353,000,000, it is estimated that 100,000,000 cases of malaria occur annually, and in only 8,000,000 to 10,000,000 of these is treatment given. Three million or more deaths from malaria are reported annually.

In British colonial territories, with a population of 63,000,000, 6,500,000 persons were treated for malaria in 1935.[(1)]

History of Treatment.

Malaria was known in Greece and Italy two thousand years ago, and Hippocrates (460 to 370 B.C.) noted the existence of quotidian, tertian, quartan and subtertian types of fever. But very little of interest from the point of view of treatment is found in the literature until the seventeenth century. From that time the history of treatment becomes divided into two periods: (i) the quinine period from 1640 till 1927, during which time quinine was the only specific drug known; (ii) the synthetic antimalarial drug period since 1927.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on October 28, 1943.

The Quinine Period.

You all know the story of how the Countess D'El Chinchon, wife of the Viceroy of Peru, was cured of malaria by treatment with extracts of the bark of a certain tree, how this bark was introduced into Europe in 1640, and how the first "h" was left out of her name and the bark came to be known as "Cinchona". It seems a pity that such a romantic story should, in the light of recent research, be found to be no more than a fable.[(2)] The real facts are these: Linnæus (1742) was responsible for the name "cinchona"; it was derived from the Indian word for bark—"*quina-quina*". Later it was found that there were two different kinds of bark—one that of the Peruvian balsam tree, and the other a similar bark of another tree, with which traders used to adulterate the (then) valuable Peruvian bark. This latter adulterant was what is now known as cinchona bark. Ignorance as to the identity of the two trees from which the bark was derived had widespread effects in causing the controversies as to the efficiency of the drug which raged during the next hundred years. In their attempts to cure malaria some were using Peruvian bark and others the real cinchona bark. By the first quarter of the eighteenth century, the place of cinchona in *materia medica* was established. The discovery of this specific drug proved a blessing to untold millions. It also enabled the malarial fevers, which were cured by cinchona, to be differentiated from fevers which were not affected by it—a diagnostic method which we, at times, are still forced to use, even today. In 1820 alkaloid quinine was isolated, and from then for many years the other alkaloids of cinchona were neglected.

Thus we have, in quinine, the case of a drug which was used for 250 years before the cause of the disease and its mode of transmission became known. Many advocates of

treatment by cinchona in the old days, including Sir Robert Talbor in England, who had some marked success with its use, were subjects of vitriolic attacks by other members of their profession. James Lind and Edward Hare in India did great work; but there was an interval of about thirty years between these two great workers when James Johnson was responsible for the abandonment of the use of the drug in favour of the "therapeutic barbarity" (Field) of his own purging and blood-letting treatment.

The Cinchona Alkaloids.—From the bark of various species of cinchona more than twenty alkaloids have been isolated; but of these only four (quinine, quinidine, cinchonine and cinchonidine) are of importance. Of these, the salts of quinine are the form in universal use. In some parts of the world the necessity for supplying drugs for mass treatment at the lowest possible cost has led to the introduction of the use of extracts of bark, without complete separation and isolation of the alkaloids. A preparation of the total alkaloids, totaquina, has been standardized by the Malaria Commission of the Health Organization of the League of Nations, and has been widely used in India since its adoption into the British Pharmacopœia in 1933. According to the new standard, totaquina must contain 70% of crystalline alkaloids, of which 15% must be quinine; in the United States Pharmacopœia the figure is 7% to 10%.

The Synthetic Antimalarial Drug Period Since 1927.

Owing to the capture by the Japanese of areas formerly producing more than 90% of the world's quinine supply, we have been forced to turn to the synthetic compounds. The introduction of these, equal (and in some respects superior) in antimalarial properties to those of quinine, is one of the greatest triumphs of synthetic chemotherapeutic research in the field of antimalarial therapy since the introduction of quinine, and it must be a bitter pill to the Germans to know that the credit for this achievement belongs to their own scientists, who introduced "Plasmochine" in 1927 and "Atabrine" in 1933. In spite of the synthesis of thousands of similar compounds by chemists since then, none has yet proved superior to these two. What would have happened to us without them is too horrible to contemplate. In the absence of specific drugs in a hyperendemic malarial area, one sees infection on top of infection, recurrence after recurrence, and (in all those subjects who do not quickly acquire a certain amount of immunity) anæmia, cachexia and death. I saw such a condition recently, when supplies of antimalarial drugs were so short that only those with threatened cerebral malaria were given treatment. There were about 30 deaths among 200 men, and the survivors when rescued were absolute physical wrecks.

After the years of practically static treatment of malaria by quinine, two factors were responsible for great strides in the knowledge of the disease: (i) the new synthetic drugs, and (ii) the induction of malaria in the treatment of general paralysis of the insane. The arsenicals "Stovarsol" and "Salvarsan" are also to a certain extent antimalarial, being mainly effective against *Plasmodium vivax*.

There is a similarity in chemical structure between all the present malarial remedies: (i) the arsenicals are built up from the benzene ring; (ii) a benzene and a pyridine ring, joined, form a quinoline ring on which all the quinine alkaloids and all the "Plasmoquine" types are built; (iii) a pyridine ring with a benzene ring on each side forms the acridine ring, on which all the "Atebrin" types of drugs are built.

Action of Antimalarial Drugs.

As it is now realized that the known antimalarial drugs differ in their effect on successive developmental stages of the various types of malaria, the action of the drugs will be described in relation to these stages: (i) action on the sporozoites, (ii) action on the trophozoites, (iii) action on the gametocytes.

Action on the Sporozoites.

Practically nothing is known as to what happens to the sporozoites (the progeny of the sexual cycle of the *Plasmodium* in the mosquito) during the ten days or so from the time they are inoculated into the human body by the mosquito, until the trophozoites appear in the blood. They just disappear, and there is no known drug which in non-toxic doses will destroy them. ("Plasmoquine" in toxic doses has been successful in preventing the appearance of the trophozoite stage.)

Action on Trophozoites.

The trophozoite stage is that stage in the human asexual cycle, against which all the present remedies for cure of an attack of malaria are directed, from the first small ring form in the red corpuscle to the stage of sporulation, when the merozoites are thrown out of the burst corpuscle to enter fresh corpuscles and repeat the process. Quinine and "Atebrin" both have a pronounced destructive action on this stage of all forms of malaria. "Plasmoquine" also destroys these asexual forms of *Plasmodium vivax* (benign tertian malaria), but not those of *Plasmodium falciparum* (subtertian malaria).

Action on Gametocytes.

Some of the trophozoites, instead of proceeding to the sporulation stage, develop into male and female gametocytes, and have no further effect, so far as the disease is concerned, on the individual. They simply remain in the blood, waiting to be ingested by a female anopheline, and so go on to the sexual half of their life cycle in the mosquito. Thus drugs which destroy this gametocyte stage in the human are administered solely to prevent the infection of fresh mosquitoes, and thus to limit the propagation of the disease. Quinine and "Atebrin" are gametocides in benign tertian malaria, but neither has any effect on the gametocytes (crescents) of subtertian malaria. "Plasmoquine", however, has a very potent effect on the gametocytes of subtertian malaria, as well as on those of benign tertian malaria.

Comment.

Thus, quinine or "Atebrin" alone will destroy both trophozoites and gametocytes of benign tertian malaria; but for subtertian malaria, quinine or "Atebrin" is necessary for the trophozoite stage, and "Plasmoquine" to destroy the gametocytes. It must also be remembered that different strains of the same species do not always respond in the same degree to the same drug. No drug is yet known which will destroy all stages of human malaria parasites.

"Atebrin" Type Drugs.

"Atebrin" type drugs include "Quinacrine" (French), "Mepacrine" (English), "Acriquine" (Russian). "Atebrin" type drugs are highly active against the asexual forms of all parasites. Their action relative to quinine differs with different strains of parasites. For *Plasmodium falciparum*, quinine is more rapid in New Guinea and Malaya; but "Atebrin" is better for the European strain. For *Plasmodium vivax*, "Atebrin" is stated to be more rapid in action than quinine (see the fourth annual report of the Malaria Commission of the League of Nations).[3]

Relapse Rates.

Relapse rates similarly vary with different strains in different parts of the world. The slow excretion of "Atebrin" compared to that of quinine has largely influenced the findings.

"Quino-Plasmoquine."

Owing to the absence of schizonticide action of "Plasmoquine" in subtertian malaria, it has been combined with quinine as "Quino-Plasmoquine" ("Plasmoquine" 0·01 gramme, quinine 0·3 gramme). The use of this combination will be discussed later.

Toxicity of the Drugs.

Quinine.—Quinine is the least toxic of all the antimalarial drugs. Cinchonism causes ringing in the ears, dizziness, temporary deafness and sometimes nausea. Rare cases of supersensitiveness occur. An unusual case of quinine idiosyncrasy was encountered in Port Moresby.

> An Australian soldier was admitted to the base hospital suffering from obvious malaria. He informed the medical officer that he was unable to take quinine, as it was a "poison for him". On being questioned as to the reason for his statement, he reluctantly explained that when his wife used quinine pessaries, he invariably developed itching skin patches and severe œdema of the genitals. He agreed to a quinine scratch test, which immediately produced the urticaria and œdema he had described.

"Plasmoquine."—"Plasmoquine" is a toxic drug, and the dosage suggested in the original package (0·02 gramme three times a day) caused such alarming symptoms that its use was discontinued in New Guinea for some years. The toxic effects are: (i) the formation of methæmoglobin, with resulting pronounced cyanosis, (ii) severe abdominal pain, (iii) an adverse effect on the circulatory system.

"Atebrin."—"Atebrin" is relatively harmless for most subjects. As a rule it causes much less discomfort than quinine medication. Towards the end of a course, sometimes abdominal discomfort and pain occur. Staining of the skin takes place in a certain proportion of cases; this is in no way relative to jaundice. Hecht states that very little "Atebrin" reaches the general circulation until the liver has been saturated. This is interesting, as in New Guinea it was found that less skin staining occurred if glucose (or barley sugar) was taken before and during the course of "Atebrin", and also that there were fewer cases of abdominal pain.

Occasionally patients with idiosyncrasy are found, who are violently ill after a single dose, and cases of cerebral irritation have been reported (the "Atebrin" psychosis of Malaya). I have never seen anything more severe than restlessness and excitement. "Atebrin" and "Plasmoquine" should never be given simultaneously—the combined toxicity is greater than that of either drug given alone. Fatalities have been reported. "Atebrin-musonate" ("Atebrin" for injection) is a soluble salt, which is stated to be dangerous to use intravenously. Its use by intramuscular injection will be discussed later.

Other Drugs.

Although quinine and "Atebrin" effectively control the schizonts of malarial parasites, and although "Plasmoquine" is equally effective as a gametocide in *Plasmodium falciparum* infection, none of them can be regarded as a true chemo-prophylactic agent. The ideal antimalarial drug has still to be found. Systematic investigations of the many organic sulphur compounds are now being made, but no definite conclusion has yet been reached. Not only have favourable clinical results been reported on the use of sulphonamide compounds in all three forms of malaria, but sulphapyridine has been shown to destroy the sexual as well as the asexual forms of *Plasmodium vivax*, and it has been found that no relapses occur after large doses. Future work will probably be directed towards the finding of a new compound with true prophylactic properties, which are not possessed by quinine, "Atebrin", "Plasmoquine", or any combination of them.

Immunity.

As an infectious disease, malaria may be looked on as a particular type of disseminative infection, such as pyæmia, typhoid fever or plague, but the bacteria of the last-mentioned are carried in the blood plasma, while the plasmodia of the former are in the red blood corpuscles. In hyperendemic areas, we see the inhabitants, with multiplicity of infections, acquiring a relative and partial immunity. In these areas, such as those of the coastal regions of New Guinea, the infant mortality averages 25%, of which it is estimated 20% is due to malaria. Every infant contracts malaria, and only the fittest survive. By the time adult life is reached, immunity has developed, as shown by the following facts. (i) Practically all deaths from untreated malaria in hyperendemic countries occur in infancy and childhood. (ii) Adults recover in the absence of antimalarial drugs. (iii) Adults suffering from fever and having parasites in their blood are quickly cured of their attack by very small doses of quinine. (iv) Cerebral malaria, although common in native children, is rare in adults. (v) Parasite surveys reveal a much higher parasite (including gametocyte) rate in children than in adults.

But the immunity of these people has always been found to be partial, and not total, no matter how great was the multiplicity of infections. The reason for this has probably been found in experiments in the treatment of general paralysis of the insane by induced malarial infections, which have shown that immunity is developed only for the particular strain of the parasite (*Plasmodium vivax* or *falciparum*) used in the experiment.[6] The development of even this immunity was found to be interfered with by early termination of the infection by drug treatment.

The multiplicity of strains would explain the failure of native races to develop full immunity, and it also may be found to account for the "blackwater areas" noted over many years, due, I submit, to a particular strain of subtertian infection. Immunity to malaria is noticeably linked up with immunity to blackwater fever.

Treatment of Acute Attacks.

No matter what the text-book or journal or report on tropical medicine one consults, there will be found in each a scheme for the amount and duration of dosage of antimalarial drugs differing from all the others. But we now know that both benign and subtertian malaria differ greatly in virulence in different countries, in endemic as compared with hyperendemic areas, and in relation to different anopheline vectors. I am therefore, in this section on treatment, limiting my remarks to the practical application of the experiences of myself and my medical colleagues of malaria in New Guinea—a hyperendemic zone, with a single anopheline vector (*Anopheles punctulatus*). This will be followed by a discussion of recent schemes of routine therapy in America and Australia.

Treatment of Subtertian Malaria (Plasmodium Falciparum Infection).

Heavy Primary Infection.—The New Guinea strain of *Plasmodium falciparum*, in an acute attack, is better controlled by quinine than by "Atebrin". The routine treatment in New Guinea was the administration of five grains of quinine bihydrochloride in capsules every four hours for six doses per day (or ten grains three times a day) until the temperature had been normal for twenty-four hours. In all cases associated with hyperpyrexia, with signs of toxæmia from heavy infection, or with vomiting severe enough to leave doubt as to the assimilation of the quinine given orally, intramuscular injections of quinine (nine grains) or "Atebrin-musonate" (0·3 gramme) were immediately given, and repeated on the following day if vomiting persisted. After the temperature had sunk to normal, the dosage of quinine was reduced to twenty grains per day for seven days, or a five-day course of "Atebrin" (0·1 gramme three times a day) was given. A course of "Plasmoquine" (0·01 gramme three times a day) for five days was given if crescents were found in the blood after the quinine or "Atebrin" course was completed. In areas where there was little likelihood of reinfection, treatment was then stopped; but if the patient was returning to a hyperendemic area, ten grains of quinine per day were given for another fortnight, and then the routine dosage of five grains per day was given in order to reduce the severity of a reinfection, even though it retarded the development of immunity.

Milder Subtertian Infection.—In a milder subtertian infection the same oral treatment with quinine or "Atebrin" was given, or, if one wished to avoid forty-eight hours of acute discomfort, an intramuscular injection of "Atebrin-musonate" (0·3 gramme) was given, followed by a course of "Atebrin" or quinine, twenty grains per day for a week.

XUM

Recurrences.—Recurrences were treated in the same manner as milder subtertian infections. There are few recurrences of subtertian malaria in New Guinea.

"*Low Fever.*"—The term "low fever" is a common one in hyperendemic areas, although seldom referred to in text-books. It is, however, a definite condition of mild pyrexia (the temperature is seldom above 100° F.) at some time during every day, with accompanying aches and general run-down feeling. There are two types: (i) a type seen in people in fairly heavily infected areas who are being continuously reinfected and who are taking insufficient suppressive treatment (the usual five grains of quinine per day); (ii) the type seen in inhabitants who have lived there for many years, who take no suppressive treatment, but who have developed sufficient immunity to prevent acute recurrences. In the former case the fever usually disappears when the suppressive dosage is increased, but in the latter it is often refractory to quinine given orally. These patients, however, respond satisfactorily to intramuscular injections of quinine.

Cerebral Malaria.—In cerebral malaria, the intravenous administration of quinine (ten grains in ten cubic centimetres injected very slowly) is essential; this may be repeated in twelve hours if coma occurs, and may be followed by intramuscular injections of nine grains of quinine until the patient regains consciousness. In a paper on pernicious malaria in *Transactions of the Royal Society of Tropical Medicine and Hygiene*, Volume XXXVIII, July, 1943, Lieutenant-Colonel D. K. Lindsay states that 30,000 non-immune labourers were stationed in a valley inhabited by 200 natives whose "spleen rate" was 100%. The annual fever rate of the labourers was 250%, and many cases of pernicious malaria occurred. The following types were noted: (i) algid, requiring immediate urgent treatment; (ii) cerebral; (iii) hæmorrhagic, with epistaxis, and rectal and petechial hæmorrhage; (iv) gastro-intestinal. Lindsay saw 10,000 intravenous injections given, with no untoward effects. In the same area, 2,000 intramuscular injections were given annually, with only one abscess.

Lindsay suggested that if a patient having a rigor was deprived of blankets, he would not sweat.

Lumbar puncture is necessary in cerebral cases for diagnostic purposes as well as for decompression. For children one must use intramuscular injections of quinine (not "Atebrin"). Children stand more than the usual percentage of age dosage commonly used for other drugs.

Treatment of Benign Tertian Malaria (Plasmodium Vivax).

Acute Primary Infections.—Acute primary infections of benign tertian malaria in New Guinea were seen mainly in the settlements and not in the bush. They are capable of control along lines of treatment similar to that for less severe subtertian malaria. Intramuscular injections of "Atebrin-musonate", if the attack is severe and accompanied by vomiting, followed by quinine or "Atebrin" as in the milder forms of subtertian malaria, are sufficient.

Chronic Recurring Benign Tertian Malaria.—The New Guinea strain of *Plasmodium vivax* seems to be exceptionally stubborn. There are recurrences in some cases in spite of (*a*) repeated full courses of quinine or "Atebrin", (*b*) short treatment of recurrences, (*c*) the use of "Quino-Plasmoquine", (*d*) intramuscular injections and (*e*) "Salvarsan" given intravenously. I have had two patients refractory over a period of one or two years to all the foregoing treatment. Each was finally cured by forcing the development of immunity, by withholding all treatment until the advent of the second or third forty-eight hour rigor, and then giving only sufficient quinine to bring the temperature to normal (only 20 or 30 grains). In the more severe of the two cases, three attacks were allowed to go to the second severe rigor, and the fourth to the third, before quinine was given; but that was the last recurrence, and there have been no more during the last two and a half years. Repeated full courses of treatment with quinine, "Atebrin" and "Plasmoquine" in these chronic recurring infections not only waste drugs, but their failure to prevent recurrences depresses the patient. It is much better to tell him that recurrences are inevitable, that he is to have no treatment between attacks, and that if he takes just sufficient quinine (or "Atebrin") to allay the attack, the following one will be milder, the next milder still, and so on. With each attack he is building up an immunity for permanent cure and future protection. Again, daily doses of five grains of quinine after one leaves an infected area only put off the evil day of recurrence.

The following case is typical:

> A man and his wife, who had both had benign tertian malaria in New Guinea, went on a holiday to America. Each took regularly five grains of quinine per day for three months, and at that stage the quinine supply ran out. Ten days after ceasing to take quinine, they both developed severe recurrences of benign tertian malaria.

Acute Benign Tertian Malaria in Children.—In heavy infections in children, early drastic treatment is essential, and the intramuscular administration of quinine is often necessary. Benign tertian malaria in children is likely to become rapidly serious, and alarming hyperpyexia develops. I have seen two fatal infections in young European children, in which adequate treatment was commenced too late. Each child had a temperature of 106° F. before death.

Intramuscular and Intravenous Therapy.

Quinine.

All authorities agree as to the necessity for the intravenous injection of quinine as soon as possible in cerebral malaria, especially if the patient is comatose when admitted to hospital. But there is a divergence of opinion regarding the intramuscular administration of quinine.

In a recent address to the Royal Society of Tropical Medicine and Hygiene on the treatment of malaria, the President, Sir Rickard Christophers (*Transactions of the Royal Society of Tropical Medicine and Hygiene*, August 31, 1942) spoke strongly against the intramuscular use of quinine in "cerebral or other pernicious manifestations", and even suggested "medical malpractice". In the discussion which followed, this statement was challenged. The first speaker, Dr. William Hughes, of Nigeria, said: "I only know that intramuscular quinine, plus saline and glucose intravenously, is remarkably successful." The next speaker, Professor Warrington Yorke, of the Liverpool School of Tropical Medicine, said: "It was with surprise and consternation that I listened to Sir Rickard's scathing denunciation of the intramuscular use of quinine. In Liverpool, where we must have given many hundreds of intramuscular injections during the last twenty-five years, we have never produced the dire results referred to, and I am confident we have saved many lives." The next speaker, Major T. R. Hill, said: "Intravenous is more effective than intramuscular quinine, and the latter should never be given." The last speaker, Sir Philip Manson-Bahr, said: "There has been a lot of loose talk about intramuscular quinine. I have always said, and maintain now, that one or two injections work miracles in very serious cases."

I myself have not seen a single abscess in hundreds of cases in which quinine was given by the intramuscular route during the last twenty years. If the injection is given deeply into the muscle in the upper and outer quadrant of the buttock, and if the buttock is well massaged for a few minutes, tenderness for a week or so is the only "dire result".

The intramuscular administration of "Atebrin-musonate" does not establish control so quickly as does quinine given into the muscle; but tenderness is decidedly less in intensity and in duration.

Experience in New Guinea is exactly in line with the findings of J. W. Field in Malaya ("Notes on the Chemotherapy of Malaria", 1938, page 116) who summarizes the position as follows: Injections are indicated: (i) when antimalarial drugs cannot be swallowed; (ii) when their retention is doubtful (owing to vomiting); (iii) when absorption is doubtful; (iv) when many parasites are found (heavy infection); (v) when the patient's condition

is clinically grave. To these patients, with the exception only of those with coma or threatened coma, quinine or "Atebrin" should be given by intramuscular injection. "Atebrin" should never be given intravenously. In malarial coma, a single intravenous injection of quinine should be given and supplemented by intramuscular injection, repeated if necessary.

Treatment in Other Countries.

United States of America.

Weed, in an article entitled "The Critical Antimalarial Problem and its Solution",[5] deals with the loss of Java quinine, the restriction of the use of quinine to treatment, and the use of substitutes.

The Subcommittee on Tropical Diseases of the National Research Council (United States of America, 1943) recommends the following alternative lines of treatment: (i) combined quinine, "Atebrin" and "Plasmoquine" treatment (method of choice): (*a*) "Totaquine" or quinine, ten grains three times a day for two or three days or until the fever is controlled, (*b*) "Atebrin", 0·1 gramme three times a day for five days, (*c*) two days without treatment, (*d*) "Plasmoquine", 0·01 gramme three times a day for five days; (ii) "Atebrin" and "Plasmoquine" treatment in simple *Plasmodium vivax* infections: (*a*) "Atebrin", 0·1 gramme three times a day for seven days, (*b*) two days' rest, (*c*) "Plasmoquine", 0·01 gramme three times a day for five days; (iii) quinine and "Plasmoquine" treatment: (*a*) quinine (or "Totaquine"), ten grains three times a day for seven days, (*b*) "Plasmoquine", 0·01 gramme three times a day during the last five days of (*a*); (iv) suppressive treatment: "Atebrin", 0·1 gramme twice a day after food, twice a week.

Treatment in the British Army.

For British soldiers on active service, whose physical exertion is more severe than that of the ordinary European soldier, early treatment and large dosage have been found necessary.

Major T. Rowland Hill (British Army malaria consultant) states that he obtained quicker cures and fewer relapses by the use of combined quinine and "Atebrin". Ten grains of quinine were given three times a day and 0·1 gramme of "Atebrin" three times a day for seven days; this was followed after two days' rest by 0·01 gramme of "Plasmoquine" with ten grains of quinine three times a day for five days. As an anti-relapse treatment, Hill advocates 0·1 gramme of "Atebrin" per day.

Standard Treatment in Australia.

The standard treatment of malaria in Australia is the administration of ten grains of quinine sulphate three times a day for three days, followed by 0·1 gramme of "Atebrin" three times a day after food for five days, followed again by 0·01 gramme of "Plasmoquine" three times a day after food, and five grains of quinine sulphate three times a day after food for five days. If it is more convenient, quinine can be given in a single dose at night.

The course of "Plasmoquine" and quinine treatment may be reduced from five to three days, (*a*) in uncontrolled areas of hyperendemic malaria, and (*b*) where, for military reasons, it is necessary to shorten the course of treatment.

After conclusion of the standard therapeutic course of treatment, every patient will continue to take one tablet of "Atebrin" (0·1 gramme) on six days each week for six weeks.

From the foregoing it will be seen that in all parts of the world schemes of treatment are gradually becoming more uniform, awaiting a new and better antimalarial drug.

General Treatment.

Specific drug treatment in malaria is all-important; but there are a few points in general treatment which need mention.

1. Diet should be light while the temperature is raised, and before a paroxysm is due.

2. Hot lemon drinks and aspirin during the shivering stage partly relieve the severe headaches.

3. Pyjamas and bedding should be changed after sweats.

4. Plenty of liquids should be given.

5. Rest in bed is required during the febrile period, but not for the whole duration of the course of treatment. Malaria patients in their first few attacks tend to be very sorry for themselves, and to keep them in bed after their fever has been controlled is very likely to render them introspective and morbid about their condition.

During convalescence, iron and arsenic tonics are necessary. Large doses of *Ferri Carbonas Saccharatus* with pooled *A*, *B*, *C* and *D* vitamins are useful for the anæmia. "Novarsenobillon" (0·6 gramme) given intravenously is useful in chronic malarial debility, especially that associated with chronic benign tertian malaria. Men who have lived in the bush for years and have developed a partial immunity similar to that acquired by natives, come in every twelve months or so and ask for their "shoot" of "Novarsenobillon".

Conclusion.

In conclusion, I quote Dr. Nieve Kingsbury, Director of the Institute for Medical Research, Kuala Lumpur, Federated Malay States: "Dogmatism can find little place in malariology, and particularly is this the case in regard to Therapeutics; for even strains of the same Plasmodium may show different responses. Dosages and schemes of treatment, therefore, cannot always have universal application."

References.[1]

(1) R. P. Strong: "Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases", 1942.

(2) A. W. Haggis: "Fundamental Errors in the Early History of Cinchona", *Bulletin of the History of Medicine*, Volume X, 1941, pages 417, 568.

(3) Fourth General Report of the Malaria Commission: "The Treatment of Malaria, Study of Synthetic Drugs, as Compared with Quinine in the Therapeutics and Prophylaxis of Malaria", *Bulletin of the Health Organisation of the League of Nations*, Volume VI, December, 1937, page 897.

(4) American Association for the Advancement of Science: "Human Malaria", 1941.

(5) L. H. Weed: "The Critical Antimalarial Problem and its Solution", *Tropical Diseases Bulletin*, Volume XLI, Number 5, 1943, page 361.

THE TREATMENT OF COMPOUND FRACTURES OF THE FEMUR IN BATTLE CASUALTIES AT A GENERAL HOSPITAL.

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THE following three methods of treatment of compound fractures of the femur are advocated: (i) slinging of the injured limb in a Thomas slint, with knee flexion piece attached, and with extension applied by means of skeletal traction by weights; (ii) immobilization of the injured limb in an ordinary Thomas bed knee splint, with fixed extension by adhesive strapping applied to the skin; (iii) immobilization of the injured limb in a single or double plaster of Paris spica of the hip.

The surgeon must be prepared to use any one of these three methods according to the type of fracture. In this series the first method was most frequently used and was found to be the most comfortable. It also offered the greatest facilities for nursing the patient, and in the treatment of compound fractures of the femur the nursing is of paramount importance.

The second method is advocated in the following circumstances: (*a*) when there are wounds at the site where the Steinmann pin would have to be inserted—that is, at the upper end of the tibia; (*b*) in some transverse

[1] Acknowledgement is made specially to J. W. Field's "Notes on the Chemotherapy of Malaria", 1938.

types of fracture, which are more easily controlled by fixed extension. In these cases a slight degree of over-extension for the first day or two allows a better apposition to be obtained by adjustment of the slings, and then the extension is slackened to allow locking of the fragments.

The third method is advocated in the following circumstances: (*a*) when considerable loss of bone from the femoral shaft has occurred; in these cases the limb must be allowed to shorten so that the fragments are in contact in order to obtain union; the spica then is the only way to obtain this result; (*b*) in the later part of the treatment, when the hip joint is involved and ankylosis is inevitable, in order to obtain sound ankylosis in optimum position.

Detailed Description of the Three Methods of Treatment.

The three methods will now be described in detail.

The First Method.

Upon his arrival at hospital, the patient is given time to recover from the effects of the journey. He is then taken to the operating theatre and given a general anæsthetic. The upper half of the spica or Tobruk plaster in which he has travelled is removed, and if possible, the Steinmann pin is inserted prior to the removal of the posterior shell of the spica or the Thomas splint, in order to prevent movement of the fragments.

A Steinmann pin is preferred to Kirschner wire for the following reasons.

1. When a Steinmann pin is driven through the bone it acquires a firm grip. The pin never moves in the bone, owing to this firm grip and to the revolving collar attachment on the Böhler stirrup used in conjunction with the pin. It is found at the end of ten or more weeks that the pin is just as firm and difficult to extract as the day it was put in, and there is no ulceration of the skin at the point of entry or exit.
2. The Böhler stirrup, though light, does not vibrate and move if the splint is knocked.
3. A Steinmann pin never breaks.
4. It can be more quickly and easily inserted without the need of a complicated instrument, which may get out of adjustment.
5. A Kirschner wire has to be inserted through a drilled hole in the bone, and the drill hole is larger than the wire; consequently the wire has no grip in the bone. With each movement of the patient the wire rotates in the bone, and this causes a predisposition to loosening of the wire and infection. It may also gradually slip sideways in the bone and thus predispose to infection.
6. The heavy Kirschner stirrup vibrates and wobbles if it or the splint is knocked.
7. It is not uncommon for a Kirschner wire to break, either during its insertion or in the course of treatment.
8. When the chocks of the Kirschner drill become worn it works uncertainly, making the insertion a slow and irritating process.
9. When the wire is removed there is frequently some ulceration of the skin, or cutting of the skin at the points of entry and exit.

The Steinmann pin should be a fine one, about three millimetres in diameter. The stirrup should be the Böhler pattern, with revolving collars on each side holding a set screw which grips the pin. These allow the stirrup to rotate without rotating the pin in the bone. The set screws must have good stout threads. In many army patterns the threads are poor. The stirrup must be large enough so that these collars are well clear of the skin—that is, about twelve to fifteen centimetres wide.

The pin is inserted through the tibial tubercle about two centimetres distal to the upper articular surface of the tibia, where it can be driven easily through cancellous bone. If it is inserted further down the shaft, the insertion is more difficult.

The stirrup is attached and an assistant can then apply and maintain manual traction while the posterior half of the spica, or the Thomas splint used for transport, is being removed.

The nature and site of the wounds are then examined. One must realize that when the limb is slung in the Thomas splint with the knee flexed, the proximal end of the thigh is the lowest part and drainage will take place in this direction. Gravitational abscesses are prevented by making sure that the wound has dependent drainage. For this reason one should not hesitate to enlarge the wound in a proximal direction or provide counter drainage by a proximal incision if necessary. Gravitational abscess will be uncommon if this point is appreciated. No search should be made for metallic foreign bodies unless these are large or one suspects that they may produce pressure on the sciatic nerve. Loose bone fragments are not removed. It has been found that sequestration of apparently loose fragments is uncommon, if sepsis is controlled. Wounds are covered, not packed, with "Vaseline" gauze and a pad of wool bandaged lightly in place.

The Thomas splint, with knee flexion piece attached, is then threaded over the limb. The ring of the splint should be about 26 or 28 inches. The ring is not to be used for counter pressure. It must be large enough to reach under the buttock and not come in contact with the anterior superior iliac spine when the patient sits up.

The slings used to support the limb must be made of firm, unyielding material such as good unbleached calico or ticking. Flannel or flannelette is useless. One sling is placed under the site of the fracture and another under the knee. The operator then attaches the cord (strong army pattern with hook attached) to the stirrup. The cord is passed around the "W" at the end of the Thomas splint, then back through the stirrup and back to the end of the Thomas splint. This gives a multiplying pulley effect, so that the extension can be tightened and the fracture easily reduced. Radiological control is not necessary at this stage. The remaining slings are then placed in position—that is, three under the thigh and three under the leg. The patient is then returned to the ward.

The bed should have fracture boards under the mattress, and the foot should be raised nine inches. In the ward, the Thomas splint is then arranged as follows. The distal end is suspended to an overhead beam by a cord. This is attached to each side bar of the splint and passes around the overhead beam. A wooden beam, rectangular in cross section, is preferable, and one complete turn of the cord around this will prevent the splint from rotating. Weight suspension for the splint is not necessary, and it involves unnecessary apparatus. As a rule, this is the only suspension of the splint which is required. Usually the ring sits satisfactorily under the buttock, and the patient soon learns to arrange the air ring comfortably. In some cases, however, the ring is partially suspended by a cord attached to it, which is passed up obliquely towards the head of the bed, to the overhead beam, with a five pound weight attached. This prevents the ring of the splint from slipping down the limb.

It is most important to instruct the patient to grasp the ring with one hand and pull it up with him when he lifts himself up by the overhead grip ring for attention to his back and use of the bedpan.

The weight traction cord passes from the stirrup distally just beneath the "W" end of the Thomas splint and over a pulley at the end of the overhead beam. This end of the beam should stand well away (about fifteen inches) from the end of the bed, so that the weight will fall clear. The weight required for compound comminuted fractures of the femur is, on the average, fifteen pounds, considerably less than is needed for simple fractures. This is on account of the comminution and muscle damage. Over-extension is to be avoided, as it tends to produce non-union and formation of sequestra. For a powerful man twenty pounds may be required, but never more than this. In cases of great comminution only ten pounds are required.

A radiological examination is made the next day, and the position is usually found to be satisfactory. Frequent alteration of the amount of extension weight should be avoided. Shortening of half an inch is of no importance so long as union is obtained and sepsis is controlled. The secret of success is to obtain satisfactory (not necessarily anatomical) position and alignment in the

beginning and then leave the fracture alone. Too much interference in all fracture work is fatal.

The following points in the nursing are of paramount importance, and if sisters are not accustomed to the nursing of fracture patients, they must be carefully instructed in these points:

The patients must be washed twice a day and their backs rubbed with *Spiritus Vini Rectificatus* (not commercial spirit); this must be followed by an application of starch powder. In addition, the backs are attended to twice a day again—that is, four times a day in all. Bed sores will never occur under this *régime*.

Slings must be carefully adjusted every day so that the limb is evenly supported, and the skin under them must be rubbed carefully and powdered. Two slings are especially important—(*a*) the master sling under the fracture and (*b*) the most distal sling.

The commonest error in the treatment of fractures of the femur is to allow backward angulation at the site of fracture, and this is due to the master sling becoming slack. As has been previously mentioned, the master sling must be of firm, unyielding material—that is, good unbleached calico or ticking. In compound fractures from battle casualties the wound is frequently on the posterior aspect of the thigh, and thus two rules must be observed: (i) release only one sling at a time; (ii) an intelligent assistant must support the fracture whilst the dressing is being done. After the dressing has been done, the sling must be firmly tightened.

The most distal sling must not be placed under the heel, and it must not come lower than four inches from the sole of the foot, otherwise pressure sores will occur over the *tendo Achillis*.

Foot drop is prevented by the use of a footpiece and sling. In my opinion this is far preferable to the use of strapping on the sole and suspension by a weight. The patient is encouraged to move the foot and toes from the commencement, and the sling on the footpiece must not be tight. The foot is well rubbed with lanoline once a week. Under this *régime*, claw foot or stiffness has never been seen to occur. Special attention is needed when the sciatic nerve is involved.

Dressings are done as infrequently as possible. It is realized that the most harmful thing one can do to a wound is to "dress" it. To paraphrase the dictum of Ambroise Paré: "I dressed the wound, and in spite of this, God healed the patient." This is borne out in the closed plaster treatment of wounds and compound fractures, in which, although the wound is infrequently dressed, it heals miraculously. If there is not much discharge, the wound is dressed only every third or fourth day. It is of no moment if the slings become soiled. They are easily changed. Two things are necessary in the treatment of wounds and compound fractures: (i) drainage and (ii) rest. When this is realized, compound fractures will cease to be a problem.

Patients are encouraged to sit up and move around in the bed. At the end of five weeks, they commence contractions of the quadriceps muscle and some movements at the knee. This apparently does no harm to the fracture and appears to help union. At any rate, the patients who move about seem to obtain union quicker than those who do not. Passive movements of the patella by a masseuse do not seem to be necessary. Most fractures are sufficiently united in ten weeks for the pin to be removed and a calliper to be fitted. Union is judged by the following criteria: (i) callus showing in the X-ray film (beware of this, as the camera may lie); (ii) palpation of the fracture site with the weight removed (it should feel quite solid and the callus should not be tender).

The calliper should be a good one, and when it is fitted the most important thing is to regain knee movement. The calliper must be removed at least three times a day for this purpose, otherwise it becomes a snare and a delusion and may lead to delay in the regaining of knee movements. Movement of the hip joint and development of the gluteal muscles must not be neglected.

The Second Method.

The second method of treatment of fractures of the femur, originally designed by Hugh Owen Thomas, has been used ever since by the Liverpool school, and it gives results unsurpassed by other methods. Its disadvantage is that it requires great attention to detail in nursing, which is often difficult to attain. In this method, the ring of the Thomas splint has to take the counter pressure against the ischial tuberosity, and this demands a well-shaped and well-fitting splint, often difficult to obtain in the army. The ring should press against the ischial tuberosity without exerting undue pressure on the origin of the adductor muscles and without any pressure at all on the anterior superior iliac spine or skin just below and lateral to this. If, however, the ring is ill-fitting (as it frequently is), pressure on these points can be easily overcome, either by raising the foot of the bed nine inches and tying the distal end of the splint to the bed rail, or by using ten pounds of weight traction tied to the distal end of the splint. The latter method, with the splint suspended in addition, allows the patient more movement in bed.

Extension is applied by means of one way stretch "Elastoplast", a strip being placed along either side of the leg with no encircling strips. This strapping is bandaged on with a gauze bandage. With a little ingenuity the wounds can be avoided. The distal ends should come down to the level of the ankle joint, and they are tied or stitched to tape, lamp wick or cord. The outer cord is then passed over, and the inner cord under, the side bar, and they are tied together over the "W" at the distal end of the splint.

Whilst reduction is being obtained the extension strappings must be frequently tightened, every two hours in some cases. One must remember that by this method, especially in comminuted fractures, it is easy to over-extend the fragments. Measurements of the length of the injured limb should be compared at frequent intervals with those of the sound one. Control by examination with X rays should also be used.

It is an axiom that two-thirds of the limb must lie anterior to the side bars of the Thomas splint. Care must be taken to see that the foot is not allowed to drop, through sagging or loosening of the slings under the leg. If this occurs, the strapping extensions will cut into the back of the lower part of the leg and produce pressure sores.

As has been stated already, great care is required in nursing, and this applies particularly at the pressure points around the ring. Prior to the application of the splint, the leather of the ring should be rubbed with castor oil to soften it and render it waterproof. Where the ring presses, the skin must be attended to frequently, even every two hours in the first few days. Later the patient can be taught to do this himself, and in addition pull a different piece of skin through the ring so that the pressure is not continuous on any one area of the skin.

The Third Method.

As has previously been stated, a few compound fractures of the femur from war wounds are better treated in plaster of Paris spica casts; but these must be used with great care. If a closed plaster cast is used, soakage around the perineum must be watched for, as this will lead to bed sores and possibly result in secondary infection of the wound from the perineum. If there is much discharge from the wound, a "window" is required, otherwise the plaster cast softens too soon and has to be changed. This is only a small matter in injuries of the leg, but it involves a considerable expenditure of time and labour where a spica is concerned. A "window" does no harm if made three or four weeks after the injury, especially if the "Vaseline" gauze in immediate proximity to the wound is not changed, but only the outside pads. Little need be said about the actual application of the plaster spica. An orthopædic table and radiological facilities for controlling reduction are essential. The fracture will shorten in the spica, but this is what is aimed at in these cases. When the neck of the femur is involved, the development of *coxa vara* deformity must be guarded against.

As regards the nursing of patients in plaster spicas, it is essential that the patient be turned on to his face and nursed in the prone position for several hours per day. This allows thorough inspection and rubbing of the back and prevents lung complications and the formation of renal calculi, as well as adding to the well-being of the patient. It also aids circulation in the fractured limb, an essential factor for rapid bone union.

General Health of the Patient.

Finally, the general health of the patient must be considered. Frequent blood counts and hæmoglobin estimations are indicated in the early stages, and transfusions of blood must be given if necessary. A hæmoglobin value below 65% is a definite indication for blood transfusion. The patients are all given a mixture containing iron until any existing anæmia is overcome. Except during the first few days, sulphonamides are seldom required. The diet must be good and liberal, and an interested ward sister can do much to create an air of cheerfulness, which is so helpful to rapid recovery in these cases.

Summary.

1. Three methods of treating compound fractures of the femur in battle casualties are presented in detail.
2. The relative merits of the methods and indications for their use are discussed.
3. The importance of good nursing in obtaining satisfactory results is stressed.

Acknowledgements.

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ABDOMINAL DIAGNOSIS.[1]

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I wish to give an address today, not upon the art of diagnosis, but upon what we think we know about the science of abdominal diagnosis, about those exact facts from which we make a deduction.

In any progressive subject we build upon the errors of the past, and about abdominal diagnosis this is especially true. It has been said that any well-trained student, at the end of his first year in biology, now knows more about anatomy than did the greatest anatomist of the seventeenth century. It need not surprise us, then, that such a great surgeon as Moynihan still taught what we must regard now as utter errors, when, for example, he implicated the appendix as a common source of chronic abdominal symptoms. What do we know today that he lacked, that enables us to attain increased accuracy in diagnosis? Especially I think we understand more than he did about the nature of abdominal pain and the significance of tenderness in the abdominal wall.

By his time the simple faith that regarded abdominal pain and soreness as the same response to irritation or inflammation as would be expected in other parts of the body had dissolved before the facts elicited by surgery under local anæsthesia, whereby it had become established that the intestinal tract seemed in fact insensible, all of the ordinary painful stimuli being incapable of producing from it any sensation of discomfort.

To explain abdominal symptoms, MacKenzie had dealt exact diagnosis an all but mortal blow by reviving Ross's theory of referred pain—in other words, by postulating that though the gut appeared insensitive, appropriate stimuli applied to it were capable of producing a response in some other area of the body. Since there was no consensus of opinion as to the site of such pain reference, the position could easily arise where irritation of such an organ as the appendix might be regarded as an adequate explanation for such varied discomforts as right lower abdominal pain, mid-line pain or chronic indigestion. Since such symptoms as these sometimes disappeared after appendicectomy, it was scarcely to be wondered at that for many years an orgy of appendix removing could receive authoritative sanction. Yet we know now, on clinical evidence, that the "chronic appendix" is a myth, and it is because we understand better the production of abdominal symptoms that we know also why it must be a myth.

What, then, is the present position of the science of abdominal diagnosis? Firstly, can the apparently insensitive gut produce symptoms? It can, if the right stimulus is evoked. This stimulus is not pinching or pricking or cutting or inflammation—none of these is felt if its application is limited to the gut—but it is stretching, increase in tension in the smooth muscle fibres of the gut wall. The actual stimulus may be the stretching of the smooth muscle, or it may be a resulting secondary effect in its associated mesentery; but the net result is that sensory nerve endings in the base of the mesentery are stimulated (and these are probably somatic and not sympathetic nerves) and a painful response is produced.

This response is an ill-localized pain roughly centred on the middle line and possessing the well-known characteristics of "stomach-ache". It is ill-localized because it is produced deep from the surface, and this illustrates another important fact, that localizability is a property of superficial nerve endings only, or in other words, that if pain can be accurately localized it is superficial in origin.

The pain is centred on the middle line because so is the gut. The embryonic intestinal canal is a mid-line structure hanging from a dorsal mesentery, and though in the process of lengthening its coils have extended to all parts of the cœlomic cavity, its mesenteric pain production retains its embryonic position.

That the sensory nerve endings are somatic and not sympathetic is still disputed, but I have it on such authority as that of Professor Sunderland that the sympathetic origin of sensory nerves in the splanchnic system is quite unproved. Moreover, clinically the characteristic discomfort of "stomach-ache" as elicited, say, by a purgative is essentially unchanged when it reaches the lower part of the rectum, and can be felt not as a low mid-line abdominal pain, but as a perineal pain produced almost certainly by tension of the levator muscles (and thus certainly registered by somatic nerves).

Pain arising from the intestinal tract then can be elicited only by tensile stimuli, and it is felt as an ill-localized, mid-line "stomach-ache". What of the nature of lateral abdominal pains? These all possess in common a property lacking in mid-line pain—namely, that they are accurately localizable. Often a finger can be placed unhesitatingly on their position. We can deduce from this then that their origin is superficial. We believe that such pain can be produced in only two ways: (i) as a response to painful stimuli of the abdominal wall, or (ii) as a neuralgia of appropriate intercostal nerves. That is, they are all abdominal wall pains.

The abdominal wall is a typical somatic structure, with the ordinary pain response to stimuli. The only special interest it has for us is that its proximity to the intra-abdominal organs allows it to respond to certain stimuli transmitted to it from them, and especially to inflammation.

If, then, the acutely inflamed appendix, or the acutely inflamed gall-bladder, comes in contact with the abdominal wall, some of this inflammation may be transmitted to the sensitive parietal peritoneum, with the production of an inflammatory somatic pain—that is, localized pain and tenderness accurately defined.

Whether such pain and soreness are felt in the inflamed peritoneum, or in its corresponding skin segment, as postulated by Morley, is a matter of little importance. Except in the case of the diaphragm, the skin segment is exactly superficial to the peritoneal segment, and I have often demonstrated, when operating under local anæs-

[1] A post-graduate lecture delivered on April 3, 1943, at Brisbane.

thesia, that some at least of the pain is produced directly in the parietal peritoneum. The theory of Morley is chiefly interesting as an ingenious attempt to explain why diaphragmatic irritation is expressed as shoulder pain.

All other lateral pains can be grouped as intercostal nerve neuralgias. These neuralgias all have this in common, that the nerve supply to a segment of the abdominal wall is made sensitive, so that either spontaneous pain is elicited or all stimulation applied to such a segment is much accentuated. Now tenderness is elicited pain—pain elicited by pressure—so it follows that in any area where pain is due to local nervous sensitivity, tenderness is likewise present in that area. The maximum tenderness is felt where the nerve is most exposed to outside pressure—that is, at the outer edge of the rectus sheath, where the nerve trunk is uncovered during its passage from beneath the oblique muscles to reach the under surface of the rectus muscle. It is typical, then, of neuralgic tenderness that it is elicited most intensely at those points where the intercostal nerves cross the outer border of the rectus sheath.

The character of this neuralgic pain is as elsewhere in the body—a sharp, stabbing, throbbing or aching pain, accurately localized, extending over nerve segments, and thus in the lower part of the abdomen, being often felt also in the upper part of the thigh anteriorly.

What is the cause of this neuralgic pain? Though the nerves may be irritated by the direct effect of conditions like vertebral osteoarthritis or by renal or ureteral stimuli (though these are excluded from the present discussion), the main causes of nervous sensitization are two in number—namely, the local fixation of an emotional stimulation or the facilitation induced by long-continued mild stimuli, or most frequently, a combination of the two.

While the effect of chronic emotional stimulation, such as fear or anxiety, is to put the whole nervous system on edge, to lower the general threshold of appreciation of sensory stimulation, this does not result so much in the production of a universal discomfort as to bring to consciousness certain minimal stimuli of which in a more placid condition we should remain unaware. Sometimes the nervous person then becomes conscious of normal bodily processes, as, for example, the beating of the heart (palpitation) or the filling of the stomach at meals (fullness, flatulence, wind *et cetera*), and there are many more examples.

Sometimes the stimuli are less normal, but remain below the threshold until released by the combined effect of facilitation and central sensitization. A common example of such a stimulus in the right lower quadrant of the abdomen produces what I call the visceroptotic neurosis syndrome. Though the study of comparative anatomy has shown that the assumption of the erect posture necessitated a lateral (or dorsal) fixation of certain portions of the intestinal tract, yet in about 20% of human beings this lateral fixation of the ascending colon is absent. Such persons, from their time of assumption of the erect posture, are liable to an undue drag on their ileo-caecal mesentery, particularly when certain secondary supporting factors (mainly postural) are defective. But for such a drag to rise to consciousness, not only years of facilitation are needed (the condition is rarely felt in early childhood), but also the effect of the lowered sensory threshold induced by emotional stimulation is necessary. When all these factors act, the drag is felt as a typical neuralgia involving the lowest intercostal nerves, especially the eleventh. Such a pain will vary with the drag stimulus, so if you like you may call it a visceroptotic pain; but since the production of an emotional sensitization is necessary for its perception, we may equally regard it as a neurosis with a visceroptotic pattern. In other cases the neurosis possesses an arthritic pattern—that is, it is worse after rest, and tends to be eased by any factors which increase the circulation; in still others the pain tends to be constant, and is related to an anxiety fixed on the area involved.

We see, then, that lateral pain can be divided into two broad subdivisions: that of inflammatory origin, the inflammatory spread being particularly from abdominal organs, and that of neuralgic origin, in which the pain is usually of emotional origin, but takes the pattern of a local sub-threshold stimulation.

How can these lateral pains be distinguished from each other? Objectively it is difficult. Remember particularly that they are both associated with tenderness. The tenderness in the inflammatory condition is felt where the inflamed abdominal organ touches the parietal peritoneum; in the neuralgic condition it is felt at the outer edge of the rectus sheath.

But where the eleventh nerve crosses the rectus sheath is the site of McBurney's point, and yet that point was supposed to be the common site of appendiceal tenderness, and where the eighth nerve crosses the rectus sheath is the spot where Murphy's sign, said to be pathognomonic of gall-bladder inflammation, is elicited. The presumption is clear that many of the cases of tenderness which these earlier surgeons related to the appendix and gall-bladder were in fact due to a nervous sensitization.

Though the relation of muscle guarding to the tenderness favours inflammation, and though the spread of the pain from the abdominal wall to the anterior part of the thigh strongly favours neuralgia, yet the most accurate differentiation is made by the history.

At this point one essential feature demands emphasis—that inflammation has a limited time tenure. Though intra-abdominal inflammation may recur, it rarely does so at short intervals, while the inflammatory phase of any single attack can be measured in days or at least as a few weeks. And just as an inflammatory pain cannot act for too long, neither can it be too short. A pain that comes and goes in a few minutes is certainly neuralgic and not inflammatory.

Pain and tenderness, constantly present over months or years, must be neuralgic. Again, experience discloses that gut inflammation—and remember that the gall-bladder and appendix are diverticula of the gut—results in an interference with function. Even though the gut is not of itself registering pain, nausea, disinclination to eat, perhaps vomiting, may be present.

Conversely, in the neurotic subject, though the abdominal wall symptoms may appear acute and the general reaction to stimulation over-sensitive, yet the functions of the gut may well continue to be normal.

So to sum up this short and incomplete discussion on abdominal pain, we may state that abdominal diagnosis will become more accurate if one commences the analysis of pain and tenderness by deciding into which of three possible categories they must be placed. These categories are: an ill-localized central gut pain, an inflammatory pain and soreness in the abdominal wall, or a neuralgic pain and tenderness. Remember also that gut pain is produced only by tension stimuli, that inflammatory pain has a very limited time tenure, and that neuralgic pain, though basically emotional in origin, often presents the pattern of certain sub-threshold stimuli.

I told you in the beginning that a knowledge of abdominal pain will show why the "chronic appendix" must be a myth. As far as the "chronic appendix" is a symptomatic entity, it consists in the finding, in a patient with chronic abdominal symptoms, of localized tenderness in the right iliac fossa. It has been shown that gut responds to stimulation only by the production of an ill-localized median pain; the appendix of itself, therefore, cannot produce lateral soreness. Such lateral soreness must be either inflammatory in the abdominal wall or neuralgic in origin. But the chronicity of the tenderness renders an inflammatory origin impossible; therefore, by exclusion, it must be placed amongst the abdominal wall neuralgias.

Once we are armed with this knowledge, further investigation will speedily elicit the emotional factors that underlie its production, and also the type of pattern to which it is conforming. If you know no more than this about abdominal pain, at least you will know more about it than did even the surgical giants of the not-distant past, and your service to your patients will be correspondingly more valuable.

Reports of Cases.

OSTEOMYELITIS OF THE ODONTOID PROCESS OF THE AXIS (DENS OF THE EPISTROPHEUS).

By T. J. F. Frank, M.D. (Melbourne), M.R.C.P. (London), F.R.A.C.P.,
Melbourne.

Acute osteomyelitis of the vertebræ is a rare disease, its average frequency according to Frohman[1] being 1 to 545 cases in other bones. Its occurrence is probably more common than these statistics indicate, for cases are missed because of the situation and depth of the lesion in the spinal column. The site of the infection in the vertebral body, laminæ and processes is usually more significant than the part of the spinal column affected. If the focus is localized in the anterior part of the body, or on the periphery of a vertebra or lamina, the infection is more apt to follow the fascial planes and spinal muscles, with the production of better opportunities for eventual drainage of an abscess. However, some of these abscesses are situated behind the pharynx, in the mediastinum, along the psoas muscle and in the pelvis; consequently their real origin at times may be overlooked. Lesions of the spinous and transverse processes may finally result in superficial abscesses which can be easily drained. Should the disease affect the posterior part of a vertebral body or the interior surface of a lamina, meningitis or meningo-myelitis may arise through direct spread of the infection or from rupture of pus into the spinal canal; sometimes an extradural abscess results.

Osteomyelitis of the spine is usually the result of a blood-borne infection from a primary focus, which may affect one or more bones. In these cases, the spine may become involved weeks, months and occasionally years after the original focus of bone infection. Just as in osteomyelitis elsewhere, the infection is hæmatogenous, whilst the organisms most commonly found are *Staphylococcus aureus*, *Staphylococcus albus*, pneumococcus, streptococcus and *Bacillus typhosus*. In staphylococcal lesions, the most frequent source is furunculosis or a mild hair follicle skin infection. Other foci of infection may be infected minor wounds, impetigo, blisters or burns, and pustules of chickenpox, smallpox and vaccination which have become secondarily affected. At times no primary focus can be demonstrated; this may be due to a symptomless primary infection or to its rapid healing without leaving a clinically recognizable trace. Lowering of general resistance is an important factor, whilst trauma may produce lesions in the vicinity of an epiphyseal line, with resultant local areas of lowered resistance. The reason that organisms settle in bone and do not affect other organs is at present unknown.

Acute osteomyelitis of the vertebræ may occur at any age before their ossification is completed, and sometimes even later. Mathieu[2] regards twelve to fifteen years as the age of election and connects it with the time when secondary epiphyses make their appearance. In a study of 42 cases, Baer[3] found the localization as follows: lumbar 28, dorsal 10 and cervical 4. Other authorities have also found the lumbar part of the spine to be the most commonly involved, and suggest that this is the result of its greater mobility and consequent exposure to trauma.

Acute vertebral osteomyelitis has a high mortality, for Mathieu[2] recorded 46 deaths in 100 published cases, whilst Volkmann[4] found a death rate of 41·8% in 90 cases. Pyæmia is a common complication of acute osteomyelitis anywhere in the body, but especially in the spine; probably the abundant venous plexuses both inside and outside the vertebræ and the proximity of the *venæ cavæ* are predisposing factors. In those cases in which recovery occurs after a long period, considerable bone regeneration in the affected vertebræ and solid fusion with the adjacent vertebræ take place; Henry[5] and Kästner[6] have recorded such cases.

Reports of involvement of the cervical vertebræ were uncommon in the available literature; Makins and Abbott[7] have recorded several cases in detail. Their first was that of a boy, aged seven years, who had been ailing for fourteen days and had complained chiefly of headache; four days before his admission to hospital, a median swelling at the nape of the neck was noticed. On the twenty-first day the abscess in the neck was incised and two ounces of pus were evacuated. On the twenty-fourth day, a swelling appeared over the right greater trochanter; on the twenty-fifth and twenty-sixth days, cerebro-spinal fluid escaped from the neck wound, and on the twenty-eighth day he was delirious, lay curled up, and showed signs of irritation. Rigidity of the right arm was noticed before death occurred. *Post mortem*, the posterior arch of the atlas lay bare and white at the bottom of the abscess cavity, its two halves being separated by a median vertical fissure. An opening in the *dura mater* connected the subdural space with the abscess cavity above the centre of the arch of the atlas. Spinal meningitis, meningitis at the base of the brain, pus in the posterior cornua of the lateral ventricles, an abscess over the right greater trochanter and three abscesses in the right lung were found.

Makins and Abbott[7] also reported in their paper a six-year-old boy suffering from probable osteomyelitis of the odontoid process; this case was originally described by Ballance in 1884. Symptoms were pain in the neck and the head held stiffly for four days. On examination of the patient, the head and neck moved only with other parts of the body, the spine was rigid, whilst fullness and pronounced tenderness were present on either side of the median line in the upper part of the neck, over which the skin felt hot. The swelling gradually increased in size; on the tenth day of illness, incisions were made through brawny tissues on each side of the neck down to the transverse process of the atlas. No pus was found, but the escape of serum gave some relief. Death occurred on the fifteenth day. At autopsy, the capsules of the atlanto-occipital joints were distended with thin, fœtid pus; the synovial membranes were swollen and injected, whilst the cartilage had practically disappeared. Examination of the occipital bone and atlas revealed no disease. However, a small bare patch on the left posterior part of the odontoid process, from around which the periosteum stripped easily and left rough bare bone, and which extended a small way onto the body of the axis, suggested that the primary lesion was in this area.

Another patient recorded by Makins and Abbott,[7] a girl, aged fourteen years, complained of pain between the shoulders and in the temporal region. Swelling of the neck ensued, pronounced tenderness was present in the mid-line over the axis, and enlarged glands were palpated on both sides of the neck. Death occurred on the thirteenth day. At the post-mortem examination the whole of the axis was rough and denuded of periosteum; thick pus was present in the muscles around the bone. The periosteum of the atlas was soft and easily detached, but the bone was not bare. In addition, a large retropharyngeal abscess containing thick, dirty-white pus was found lying in front of the anterior common ligament of the first five cervical vertebræ—apparently a sequel of the osteomyelitis.

Makins and Abbott[7] also described osteomyelitis of the supraoccipital bone, a most unusual site, in a five-year-old male, and pointed out that its clinical features were similar to those of osteomyelitis of the upper cervical vertebræ. In both sites, the symptoms and signs were the result of loculation of a small quantity of pus beneath the deep muscles of the back of the neck.

Osteomyelitis in the cervical region may be mistaken for spontaneous hyperæmic dislocation of the atlas, a very rare condition. According to Dennison,[8] only 31 cases have been recorded. He described this lesion in a boy, aged five and a half years; it was noticed after an operation on the left mastoid process for acute mastoiditis, a sequel of chronic *otitis media*. The day after operation, typical torticollis without contracture or spasm of the sterno-mastoid muscle was noted; X-ray examination revealed a subluxation of the atlas on the axis, the atlas being noticeably displaced to the right side and its right side being rotated forwards. This is not a sudden lesion, for bony changes are usually found in the articular surface. In the majority of these cases the patients have been under the age of twelve years, and in practically all cases there has been an associated inflammatory change in the neck, in the naso-pharynx or near the base of the skull. Several weeks later, the child may complain of a stiff neck and resent movements of the neck, which is held in the "wry neck" position. Watson-Jones[9] has shown radiologically decalcification of the anterior arch of the atlas with complete recalcification four months later. After decalcification, the attachments of the transverse ligament of the atlas may become insecure, and with the result that the odontoid process loses its close relationship to the arch of the atlas; slight trauma may be sufficient to avulse the ligament and the atlas may be displaced forwards. Should complete dislocation result, sudden death may occur from pressure of the odontoid process on the cord. As a rule, spasm of the neck muscles prevents sub-

luxation from becoming complete dislocation; when the inflammatory process subsides, recalcification may occur. In some cases, spontaneous reduction of the dislocation has been recorded.

Trauma of the cervical vertebræ, as a rule, presents no difficulties in differential diagnosis. Jefferson[10] in 1920 collected 46 cases of fractures of the atlas, including four of his own; 21 of these were isolated fractures of the atlas and 25 were complicated with fractures of other vertebræ. Some cases have been described in which osteomyelitis supervened and the sequestrum finally separated and was expelled through the mouth after rupture of a retro-pharyngeal abscess. Syme[11] reported a case with recovery, in which the odontoid process and a large part of the body of the axis had been extruded into the pharynx. McWhorter[12] recently recorded three cases of fracture of the atlas. One of these patients, a male, aged forty-seven years, sustained fractures of the anterior and posterior arches of the atlas with a lateral displacement of the right side and a fracture of the odontoid process of the axis vertebra; the main disability was constant pain in the neck and limited movement. This patient was immobilized in a plaster cast for three months and was later able to return to his work.

Owing to the rarity of osteomyelitis of the odontoid process, it has been deemed justifiable to record the following case.

Clinical Record.

W.W.G.S., aged forty-three years, a draper, was referred to me by Dr. E. Spargo, of Merbein, and on June 20, 1941, was admitted to the Royal Melbourne Hospital for further investigation. His health had been good till three months previously, when he developed an intensely itchy swelling on the right index finger; this was followed by cellulitis of the dorsum of the hand and forearm. The lesion was attributed to a spider bite. The cellulitis subsided within four weeks after multiple incisions and the oral administration of sulphapyridine. Two weeks later, pain in the left ankle with swelling after walking was noted; this disappeared after several weeks and did not recur. Five weeks before his admission to hospital, a dull aching pain in the left loin associated with frequency of micturition and scalding occurred; this lasted four days. Three weeks before his visit, sudden neck stiffness with pain on lateral neck movements and an ache in the occipital region was experienced; he was sent to the local hospital and treated with infra-red rays and "Plastine" to the neck, with slight relief of the pain. Several days before his admission to hospital, enlarged, firm glands were noted in the left posterior cervical triangle, as well as nasal discharge, hoarseness and slight cough with expectoration of whitish sputum. Owing to the prolonged illness, the patient had become depressed. In addition, the appetite was poor and the mouth was painful; the bowels had been open regularly apart from a short bout of diarrhœa two months previously. During this illness, he had lost over thirty pounds in weight.

On examination, the patient was tall, thin, depressed and mentally sluggish. The tongue was dry, whilst the throat was reddened. Scattered rhonchi were heard in the lungs, and the urine was highly concentrated. The neck was tender over its left lateral aspect and its movements were limited in all directions; a few small glands were palpated behind the angle of the left mandible and in the left posterior cervical triangle. Routine examination of the nervous system revealed no abnormality. Lumbar scoliosis was present.

An examination of the patient's blood on the day of his admission to hospital showed that the hæmoglobin value was 103% (14·4 grammes per 100 cubic centimetres of blood), and that the leucocytes numbered 21,900 per cubic millimetre; examination of a blood film revealed no abnormality, whilst a differential leucocyte count revealed a relative and absolute increase in the number of neutrophile cells and a shift to the left in the granular series and also a corresponding relative lymphocytopenia. Of the leucocytes, neutrophile polymorphonuclear cells numbered 71·5%, old metamyelocytes 22·5%, lymphocytes 5% and monocytes 1%. An X-ray examination of the lungs on June 21 revealed slight linear fibrosis at the extreme bases and changes suggestive of a left basal pleurisy causing slight irregularity of the outline of the diaphragm. Radiological examination of the cervical vertebræ on June 23 revealed no abnormality, whilst in the lumbar region pronounced scoliosis with the convexity to the left was present. The blood serum failed to react to the Wassermann test.

On June 23 the patient complained of tender small glands in the left posterior cervical triangle associated with a swelling behind the angle of the left mandible; the throat was also sore, and greyish patches were present on the palate. The neck stiffness was still pronounced. Lumbar puncture produced a normal response to the Queckenstedt test; the cerebro-spinal fluid was under a pressure of 230 millimetres and the fluid was clear and free of globulin and contained three erythrocytes per cubic millimetre. Three days later neck stiffness was still pronounced, whilst the swelling behind the left jaw had increased in size and become tender. The appetite was fair, despite ulcers on the palate and throat; culture from a throat swab produced a growth of green streptococci, *Staphylococcus albus* and *Micrococcus catarrhalis*. On June 27 the leucocytes numbered 30,300 per cubic millimetre; neutrophilia was pronounced, and a shift to the left in the granular series and relative lymphopenia were present. The swelling behind the angle of the left mandible became larger and indurated without signs of fluctuation. The patient gradually became more ill and more drowsy, and on July 3 complained of difficulty in swallowing; on examination, complete left facial paralysis and left hypoglossal paresis were noted. Two days later, slight fluctuation was noted in the mass of glands behind the left mandible; under general anæsthesia this mass was incised deeply, but no pus was obtained. During this operation, pus commenced to discharge from the left external auditory meatus; examination at a later date revealed that the ear drum was normal, and that there was a discharging sinus on the upper posterior wall of this meatus. *Staphylococcus aureus*, diphtheroids and a small number of streptococci producing slight hæmolysis were cultured from this pus.

An X-ray examination on July 7 showed that the mastoids were only moderately pneumatized; small cells and a general loss of translucency of the cells on the left side were noted. In addition, some osteoporosis of the odontoid process on its left side was found (Figure I). As a result, a diagnosis of osteomyelitis was made.

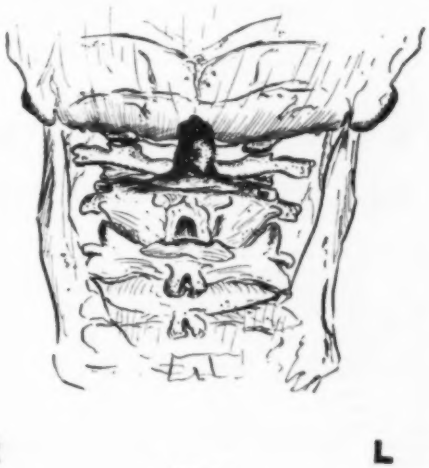

FIGURE I.

Showing osteoporosis of the left side of the odontoid process of the axis.

The cellulitis in the neck gradually subsided, leaving enlarged glands, whilst all aural discharge ceased in about ten days. On July 22 severe "toxic" diarrhœa commenced and persisted till the patient's death. Examination by a neurologist on July 25 revealed the following abnormalities: complete left facial paralysis, paralysis or absence of the left sterno-mastoid muscle (no tendon was palpable), left-sided hypoglossal weakness, and stiffness of the neck with peculiar crepitus on flexion but no definite evidence of intracranial disease or of involvement of the spinal tracts. The whole condition was regarded as due to a pyogenic infection, but in the absence of a rise in temperature, examination for actinomycosis was suggested. Further X-ray examinations of the base of the skull were advised, for the infection seemed to be in this area, possibly outside; but in view of the nerve palsies, it was thought more likely to be intracranial.

Reexamination of the cervical vertebræ by X rays on July 16 disclosed no appreciable changes in the odontoid process since July 7 and no bone absorption. Another film taken on July 28 revealed a pathological fracture of the base of the odontoid process, which was displaced about one-quarter of an inch to the right in relation to the body of the second cervical vertebra; it was also displaced a little backwards (Figure II); poor translucency of the cells of the left mastoid was still present, without any obvious destruction.

The patient gradually became weaker and complained of anorexia, diarrhœa and constant pain in the back of the neck. Lumbar puncture on August 11 produced clear and colourless cerebro-spinal fluid containing two monocytes, six polymorphonuclear cells and four lymphocytes per cubic millimetre, with no increase in globulin. Next day, the hæmoglobin value was found to be 70% and the leucocytes numbered 4,900 cells per cubic millimetre. Weakness and mental apathy gradually increased, and death occurred on August 14, 1941.

Figure II.

Showing displacement of the odontoid process to the right in relation to the body of the second cervical vertebra.

A post-mortem examination was made shortly after death and the following significant findings were noted. Faint scars were present behind the angle of the left mandible and on the dorsum of the right index finger and of the right hand.

Both lungs were large and greyish; they contained air and were emphysematous. In the upper part of the left upper lobe there was a small, thick-walled abscess containing greenish-yellow pus; the abscess did not communicate with the bronchus. The consistency of the lungs was fibrous and the bronchi were slightly dilated; the mucosa of the bronchi was congested and thick mucopus was present in their lumina.

The peritoneal sac contained several pints of clear, yellowish fluid, mostly collected in the pelvis; the lymph glands were not enlarged. The caecum and colon were thick-walled and œdematous; the mucosa throughout was congested, and hæmorrhages and some atrophy of the folds were noted. Examination of the rectum also revealed congestion, œdema and thickening of its walls.

The perinephric tissue of both kidneys was considerably thickened and œdematous, whilst around the kidneys large collections of thick, greenish-yellow pus were present; *Staphylococcus aureus* was grown on culture. The perinephric fat was opaque, and purulent infiltration was present. Both psoas muscles formed the walls of these perinephric abscesses; but no purulent infiltration was found in the psoas muscles. The kidneys were fairly large and the capsules stripped readily; the surface was generally smooth and contained a few abscess points, some of which had burst through into the perinephric region. Examination of sections of the kidneys revealed a fairly wide cortex and medulla. In both kidneys, several irregular abscesses containing thick greenish pus were present; in some of these abscesses honeycombing was noticed, whilst in others there was a radiating appearance of opaque yellow tissue, rather suggestive of an actinomycotic lesion. Microscopic examination revealed widespread necrosis and purulent infiltration with abscess formation (Figure III). Apart from the abscesses, the renal tissue was swollen and firm and the pyramids were regular. The pelvic fat and mucosa appeared normal, whilst the ureters were small, thin-walled and clear.

Figure III.

Section of kidney showing widespread necrosis, purulent infiltration and abscess formation.

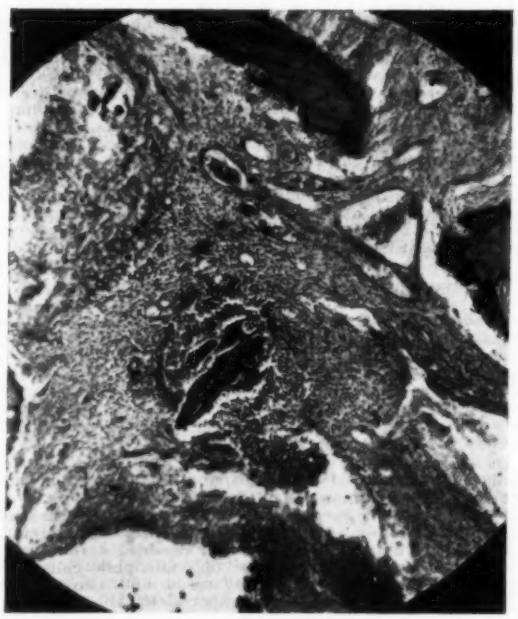

Figure IV.

Odontoid process, showing acute osteomyelitis.

Slight œdema but no cellulitis was present in the tissues at the base of the skull. The skull bones appeared normal and showed no signs of osteomyelitis. The ethmoid and sphenoid sinuses were normal. The structure of the atlas was normal. The odontoid process of the axis was congested, rarefied and eroded, and a complete fracture was present just below the level of the articular surface; this portion of the process was lying loose on the transverse ligament of the atlas and was intact. Microscopic examination of the odontoid process revealed acute osteomyelitis (Figure IV). No displacement of the atlas and axis on each other appeared to have occurred. The remainder of the vertebræ seemed to be normal.

No change was found in the cerebral *dura mater*. Clear fluid was present in the subdural space, whilst the pia-arachnoid was congested. The brain was normal in size and showed no gross lesion, although its substance was moderately congested; similar findings were noted in the cerebellum, pons and medulla. The ventricles were normal and there were no signs of compression of the brain stem.

No changes were found in the spinal dura, whilst moderate congestion only was noted in the pia-arachnoid and spinal cord.

Discussion.

The initial infection in this illness occurred five months before death and was due to a staphylococcal infection, which commenced either as a pimple or as a spider bite on the dorsum of the right index finger; as a result, swelling of the finger and cellulitis of the right hand developed. Treatment consisted of multiple incisions and sulphapyridine given orally. The infection subsided within four weeks, and was followed several weeks later by pain in the left ankle, associated with swelling on exertion, apparently the result of toxæmia.

Five weeks before the patient's admission to hospital, a dull, aching pain in the left loin and frequency of micturition and scalding were noted. Apparently, at this stage, a perinephric abscess and possibly renal abscesses or carbuncle had developed; these lesions often follow an initial staphylococcal infection after a long latent interval. As a rule, perinephric abscess causes a swinging temperature and few symptoms apart from local pain and discomfort; it must always be considered in the diagnosis of pyrexia of unknown origin. Renal carbuncle also produces pyrexia and may be diagnosed by pyelography or at operation for the drainage of some perinephric abscesses. Neither perinephric abscess nor renal carbuncle, as a rule, produces frequency of micturition or scalding; the latter symptoms may have been due in this case to toxæmia and to the high concentration of the urine. The temperature chart at no stage yielded evidence of suppuration. At the post-mortem examination, the surprising finding of bilateral perinephric abscesses was made, from which *Staphylococcus aureus* was cultured. Both kidneys contained numerous abscesses, suggestive of staphylococcal pyæmia; these lesions were old, for they were honeycombed.

Nine weeks after the onset of the illness, stiffness of the neck (sudden in onset) and pain in the occipital region with gross restriction of neck movement were noted. From subsequent radiological findings, this was the result of osteomyelitis of the odontoid process of the axis causing a reflex muscle spasm. The osteomyelitis resulted from a hæmatogenous staphylococcal infection, either initially in the hand, or more probably from the perinephric suppuration. The lymphatic drainage of the inflamed tissues resulting from the infected odontoid process apparently produced the enlarged glands on the left side of the neck and later the surrounding cellulitis. About fourteen weeks after the onset of the illness, complete left facial paralysis and left hypoglossal paresis were found. These suggested that an abscess had resulted from the osteomyelitis of the odontoid process and was tracking along the base of the skull posterior to the deep fascia of the neck. The close anatomical association of the canal which transmits the hypoglossal nerve with the upper cervical vertebræ was apparently responsible for the hypoglossal weakness; the latter was either the result of direct pressure of an abscess or due to inflammation surrounding or directly affecting the sheath of the twelfth nerve. The facial nerve emerges at the stylomastoid foramen, and a complete lesion was probably the result of periostitis of the canal at its exit caused by the neighbouring infection. Shortly after these nerve lesions were noted, an attempt to drain the cellulitic area by Hilton's method resulted in an escape of pus containing staphylococci and other organisms through the external auditory meatus at its membranous attachment to the osseous meatus. It is well known that in the lateral part of the external auditory meatus the cartilage forms about three-quarters of the circumference of the tube, but at its medial end the cartilage forms merely a part of the anterior and lower boundaries of the canal. Consequently, an abscess situated posteriorly to the canal could rupture through the membranous tissues; subsequent examination disclosed a healthy ear drum and a small sinus on the upper posterior wall of the external auditory meatus. Eighteen days after the patient's admission to hospital, X-ray examination revealed osteoporosis of the odontoid process; three weeks later, there was a pathological fracture of this process with displacement to the right in relation to the body of the second cervical vertebra; these findings were confirmed at autopsy. Apart from congestion of the pia-arachnoid at autopsy, no signs of meningeal irritation were noted clinically or by examination of the cerebro-spinal fluid.

The abscess in the lung was probably due to the pyæmia at the time of development of the perinephric and renal abscesses; it did not communicate with a bronchus, and consequently there was no history of expectoration of foul sputum.

The diarrhœa, which produced so much terminal suffering, was a manifestation of the severe general toxæmia with lowered resistance; at the post-mortem examination, a thick-walled and œdematous caecum and colon with congested mucosa were found.

Death resulted from severe toxæmia and asthenia.

Summary.

1. The clinical history has been recorded of a patient who, as a sequel to a staphylococcal infection of a finger, developed renal and perinephric abscesses, acute osteomyelitis of the odontoid process of the axis with pathological fracture, a pulmonary abscess and acute colitis.

2. Acute osteomyelitis of the vertebral column, usually hæmatogenous in origin, is an uncommon disease and has a high mortality rate. Involvement of the odontoid process with a subsequent pathological fracture diagnosed radiologically and confirmed at autopsy must be extremely rare.

Acknowledgements.

I am indebted to Dr. Barbara Wood for establishing the radiological diagnosis, to Dr. Wright-Smith for the use of his post-mortem findings, to Mr. Peter Crooke for the drawings, to Mr. Guthrie for the photomicrographs, and also to members of the staff of the Royal Melbourne Hospital for assistance in the management of the patient.

References.

(1) Frohman: Quoted by W. S. Baer, *loco citato*.

(2) Mathieu: *Revue de chirurgie*, Volume XLIII, 1924, page 96; quoted by R. L. Knaggs: "The Inflammatory and Toxic Diseases of Bone", 1926, page 27.

(3) W. S. Baer: "Osteomyelitis of the Spine in Practice of Surgery", edited by Dean Lewis, 1942, Volume III, Chapter IV, Section III, 1942, page 50.

(4) Volkmann: Quoted by H. Kästner, *loco citato*.

(5) M. O. Henry: "Acute Osteomyelitis of the Spine", *The Journal of Bone and Joint Surgery*, Volume XI (New Series), 1929, page 536.

(6) H. Kästner: *"Selterne Lokalisationen der Osteomyelitis (Wirbelsäule, Schulterblatt)", Archiv für klinische Chirurgie*, Volume CLIII, 1928, page 750.

(7) G. H. Makins and F. C. Abbott: "On Acute Primary Osteomyelitis of the Vertebræ", *Annals of Surgery*, Volume XXIII, 1896, page 510.

(8) W. M. Dennison: "Spontaneous Hyperæmic Dislocation of the Atlas", *Glasgow Medical Journal*, Volume CXXXII, 1939, page 191.

(9) R. Watson-Jones: "Spontaneous Hyperæmic Dislocation of the Atlas", *Proceedings of the Royal Society of Medicine*, Volume XXV, Part I, 1931-1932, page 586.

(10) G. Jefferson: "Fracture of the Atlas Vertebra: Report of Four Cases and a Review of Those Previously Recorded", *The British Journal of Surgery*, Volume VII, 1919-1920, page 407.

(11) Syme: "Recovery after Extensive Exfoliation of Vertebra Dentata", *Edinburgh Medical, Surgical Journal*, Volume XXV, 1826, page 311; quoted by G. Jefferson, *loco citato*.

(12) G. L. McWhorter: "Fracture of the Atlas Vertebra: Report of Three Cases, one with Removal of the Posterior Arch for Neuralgia", *The Journal of Bone and Joint Surgery*, Volume XI (New Series), 1929, page 286.

FATAL BLEEDING FROM ŒSOPHAGEAL VARICOSITIES.

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Clinical Record.

SERGEANT A.A., aged thirty-nine years, was admitted to a casualty clearing station on the morning of December 5, 1943. He gave a history of having been ill for the past six or seven days, with severe headache, generalized aches and pains (particularly in the lumbar region and on the right side) and some nausea, but no vomiting.

On examination, he was febrile, and a profuse maculopapular rash was present on the trunk and extremities. The glandular fields were not enlarged and the spleen was impalpable. No malarial parasites were detected in a thick blood film. A diagnosis of dengue fever was made, since an epidemic of this disease was present among troops in the area at this time.

At about 3.30 p.m. on the same day, an orderly reported that the patient had vomited a kidney dish full of blood. On examination, he was extremely pale and restless and was vomiting bright blood at intervals of a few minutes. About two pints of little altered bright blood had been vomited initially, and he complained of a feeling of tightness in the lower left quadrant of the chest. The radial pulse was almost impalpable and the blood pressure could not be determined, either by palpation or by auscultation. The heart sounds were faint, and the apical rate was 92 per minute. No abnormality in the chest could be detected. Slight epigastric tenderness was present.

No history of previous dyspepsia could be obtained. The patient had always enjoyed good health and had had no indigestion, abdominal pain or vomiting. There was no personal or family history of any hæmorrhagic abnormality. During his three and a half years of army service he had been employed as a cook, and his meals had been ample and regular. During the past forty-eight hours he had taken no food and little fluid. On the previous morning he had taken six "A.P.C." tablets and three more on the evening of the same day. A further three had been taken on the morning of his admission to hospital, and four more tablets two hours before the onset of hæmatemesis. In each instance the tablets had been swallowed whole and taken with a small medicine glass of water only.

A transfusion of 1,000 cubic centimetres of group IV stored blood was commenced at 4.30 p.m. From this time until 11 p.m. the patient was given a total of 4,400 cubic centimetres of whole blood, with small amounts of serum and saline solution in the intervals while the bottles of blood were being changed. The transfusion might have been at a faster rate had a quantity of stored blood been available; but apart from the initial amount of one litre, all blood had to be collected from walking wounded of blood group IV. Hypodermic injections of a quarter of a grain of morphine were given every four hours.

During the transfusion the patient vomited and coughed a few ounces of bright blood at intervals of a few minutes, and on one occasion the vomiting was noticed to be in a projectile stream. Several small melena stools were passed during the evening.

His condition improved at first, and at 9 p.m. the systolic blood pressure could be recorded at 35 millimetres of mercury, though the pulse rate had risen to 140 per minute; but later his condition began to deteriorate again. He was still vomiting several ounces of blood at intervals of about a quarter of an hour and obviously failing to respond to blood transfusion. Finally, at about 11 p.m., it was decided to continue a slow drip serum transfusion for the remainder of the night. He died at 7.40 a.m. on the following day.

Post-Mortem Examination.

A post-mortem examination was made at 9 a.m. on December 6. When the peritoneal cavity was opened, the stomach was found to be greatly distended with blood, and extensive subperitoneal hæmorrhage had taken place along its lesser curvature. The remainder of the intestinal tract also contained a quantity of blood.

No abnormality was found in either the heart or lungs; the spleen was of soft consistency and enlarged to about one and a half times the normal size. No malarial parasites were detected in splenic pulp smears. No microscopic evidence of cirrhosis was detected in the liver, which appeared normal in size and consistency.

When the stomach and œsophagus were opened, the veins above the cardia were found to be dilated, and three large varicosities about one-eighth to one-quarter of an inch in diameter were present at the cardia itself. These were thrombosed, while clot and greyish sloughs were adherent to the lower ends of all three. Two small superficial erosions were present in the intervening mucosa. The mucous membrane of the fundus was intensely congested over a wide area, but elsewhere appeared normal.

Comment.

It seems noteworthy that such large varicosities of the œsophagus should be present without any apparent hepatic cirrhosis. It is suggested that the taking of uncrushed aspirin tablets with a minimum of fluid was the precipitating cause of the patient's hæmatemesis and death. The varicosities were of such size and situation that the lodging of whole tablets, or spicules from them, in the lower end of the œsophagus could well be imagined as providing the trauma necessary for their rupture and for the profuse bleeding which followed.

Acknowledgement.

Our thanks are due to the Director-General of Medical Services for permission to publish this case report.

Reviews.

INSPECTION OF FOODSTUFFS.

IN Australia there are relatively few full-time medical officers of health. On the other hand many medical men in the country are engaged in part-time health duties. They may be called upon at any time to give a pronouncement on some food substance which has been brought to their notice by the health inspector or in some other way. To such a busy medical man "Food Inspection Notes" will be found useful.[1]

This little work has been prepared by two sanitary inspectors of London. An epitome is given of the inspection of meat, both in the living animal and in the dressed carcass. Various terms are defined, the appearance of normal organs is given and the methods of distinguishing one kind of meat from another. This is followed by an account of disease conditions in meat and the recognition of staleness and putrefactive changes. A description is given of the appearances of imported meat. There follow chapters on poultry, rabbits and game and on fish and shell-fish. Then comes a section dealing with milk and milk products and another on miscellaneous foods which include canned foods, beverages, cereals, dried fruit and fresh fruit, fermented liquors, jams, vegetables and so on. The final chapters are on food poisoning and the method of taking samples.

The object of the authors has been to refresh the mind of the inspector when, perhaps, suddenly presented with a foodstuff departing from the normal. It is unfortunate, however, that the work was not revised by a medical or veterinary officer. If this had been done, we would not find it stated that fowls are susceptible to the human type of the tubercle bacillus or that milk fever is caused by a rush of blood to the udder causing anæmia of the brain. Some of the definitions in the glossary of important terms are very poor or even misleading. Some old-fashioned names are still retained. The section on food poisoning is poor. We understand that for some time now "worms" have ceased to be found in Australian currants. There are some spelling errors. The book has been prepared for English conditions, and adjustments must be made for Australian requirements. We believe that it will be found quite useful as an epitome by medical men who are required from time to time to inspect foods and whose training would detect any inaccuracy in its contents. A new edition carefully revised would be a boon to the health inspector.

[1] "Food Inspection Notes: A Handbook for Students", by H. Hill, F.R.San.I., F.S.I.A., A.M.I.S.E., and E. Dodsworth, M.R.San.I., M.S.I.A.; 1943. London: H. K. Lewis and Company, Limited. Crown 8vo, pp. 130. Price: 6s. net.

The Medical Journal of Australia

SATURDAY, MARCH 4, 1944.

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CIVILIAN DOCTORS AND THE TREATMENT OF MALARIA.

IN August, 1942, the attention of medical practitioners in Australia was directed by a leading article in this journal to the acute shortage of quinine and to the urgent need to conserve supplies that the drug might be available for treatment of malarious patients. Since that time, as far as can be gathered, medical practitioners have heeded the warning and have done what they could to discourage any suggestion that quinine might be used for any non-malarial purpose. Steps were taken by the Medical Equipment Control Committee to eliminate quinine from certain well-known pharmaceutical preparations, and useful quantities of the drug were put on one side that it might fulfil its proper destiny. The hazardous position caused by the quinine shortage has, as was predicted in August, 1942, been largely remedied by a great increase in the production of quinine substitutes, and these drugs have justified the hopes that experience suggested should be placed in them. Two facts now call for emphasis. The first is that some thousands of men and women in the services have for many months lived and worked in anything but ideal conditions in a malaria-infested country to the north of Australia, men and women who until they joined the ranks of the services had never been exposed to the risk of malarial infection. The second fact is that supplies of quinine are still short and that there is no likelihood of any substantial improvement. Readers may remember that some months ago (on June 12, 1943) the subject of malaria was discussed in these columns, and that mention was made of the possibility that soldiers on leave from New Guinea might come into the hands of civilian practitioners for treatment while they were on leave or else that soldiers might act as a reservoir of infection to civilians in places where mosquito vectors of malaria were known to exist. It will be recalled that a case of malarial infection was reported by Dr. A. H. Tebbutt as having occurred in the southern part of New South Wales, the patient being a lad, aged seventeen years, who had been in contact with a soldier returned from active service in New Guinea and very ill with malaria. Now that soldiers are being invalided from the army after service in malarial districts, and, as the authorities announce in the newspapers, soldiers are being retired from the army that they may give much needed help in the production of food, malaria becomes a still more vital subject for the civilian practitioner. For this reason the paper by Dr. E. T. Brennan published in this issue and the discussion that followed its presentation are worthy of careful attention.

Dr. Brennan's paper must, in view of the persistence of the shortage of quinine, be read with understanding and discrimination. The introduction of the synthetic compounds "Atebrin" and "Plasmoquine" was indeed a triumph of synthetic chemotherapy. And, as Dr. Brennan also points out, quinine or "Atebrin" alone will destroy both trophozoites and gametocytes of benign tertian malaria, but for subtertian malaria quinine or "Atebrin" is necessary for the trophozoite stage and "Plasmoquine" to destroy the gametocytes. He insists, however, that it must not be forgotten that different strains of the same species do not always respond in the same degree to the same drug. "No drug is yet known which will destroy all stages of human malaria parasites." It will be noted that Dr. Brennan describes a "standard treatment of malaria in Australia" which includes the administration of quinine sulphate, "Atebrin" and "Plasmoquine". This is where the civilian practitioner must adjust his ideas to the drug situation as it is today. What Dr. Brennan was trying to show was that "in all parts of the world schemes of treatment are gradually becoming more uniform", while medical science is "awaiting a new and better antimalarial drug". The quinine position is so difficult that the authorities find it impossible to liberate supplies of quinine for the treatment of relapses of benign tertian malaria in civilians. It was for this reason that in October of last year we published in this journal the details of a course of treatment recommended by the Director-General of Medical Services for the treatment of malaria in civilians. The course was as follows: "Atebrin" 0·1 gramme three times a day for five days, followed by "Plasmoquine" 0·01 gramme three times a day for five days. It was pointed out that each dose of both these drugs should be taken after food with a copious draught of water. In the event of supplies of "Plasmoquine" being short, it was stated that a dose of 0·01 gramme three times a day for three days was usually sufficient. The Director-General of Medical Services stated that this treatment was adequate to control an attack and should minimize the occurrence of relapses under the conditions in which malaria might arise in the civil population, "the only type of infection likely to occur in civilians is the benign tertian".

Another possibility arises. Now that men are being discharged from the army to industry, many of whom will no doubt come straight from heavily infected areas, it is possible that some cases of malignant tertian malaria may occur among them. If they do occur, they may be severe infections and some may even be of the cerebral type. In the ordinary course of events men are not discharged from the army unless they have been deemed healthy, and in

these circumstances are not very likely to suffer at a later date from malignant malaria. But for all that, the possibility remains; and in any case soldiers on leave may in certain circumstances become the subjects of malignant malaria. The army authorities are aware of this possibility and efforts will be made to meet the situation. It should perhaps be stated that malignant malaria is a condition which fairly quickly runs its course. In a report by a subcommittee of the League of Nations Malaria Commission published in the *Quarterly Bulletin of the Health Organization* in June, 1933 (see The Medical Journal of Australia, December 2, 1933, page 766) we read that malignant malaria in its general clinical course is an acute disease consisting of a severe attack followed usually by several milder recrudescences at short intervals. The average number of recrudescences is four. The duration of the disease as manifested by febrile attacks is seldom less than a month and seldom longer than six months. A person therefore is not likely to suffer from malignant malaria if he has been away from the tropics for more than six months. With the treatment now being used in the Australian services it has been found that recrudescence in malignant malaria is unusual. Malignant malaria calls for the exhibition of quinine which should be given preferably by intravenous injection. It must be added—and Dr. Brennan as well as those who discussed his paper made it quite clear—that some experienced practitioners of tropical medicine hold that intramuscular injection is just as good as intravenous administration. That question, however, need not detain us. The Australian Army Medical Corps authorities have found that if a solution of quinine hydrochloride is given intravenously to a patient with malignant malaria and followed up by larger doses of "Atebrin" than usual, attacks of any degree of severity may be combated with success. They have found that the less severe cases of malignant tertian malaria may be adequately treated by "Atebrin" alone from the beginning, but that larger doses than those suggested for the standard course are needed for the first few days. It may be remarked in passing that the diagnosis of malignant tertian malaria is not a simple matter to a practitioner whose experience in such diagnoses by microscopic methods is not wide, but this point is really outside our present discussion. Any arrangements that are made to assist civilian medical practitioners in regard to supplies of quinine hydrochloride for the treatment of malignant malaria will presumably be made through a civilian authority. It is hoped that before very long it will be possible to make an announcement on the subject.

Current Comment.

SULPHADIAZINE AS CAUSE AND CURE OF AGRANULOCYTOSIS.

The literature abounds with reports of cases in which agranulocytosis has followed the administration of sulphonamide drugs. It is everywhere stated that as soon as any evidence appears that the number of granular leucocytes in the blood stream is becoming less, administration of the drug shall be stopped. In order that a change of this kind shall be discovered in its early stages during sulphonamide therapy, white cell counts are generally undertaken at frequent intervals. It has been pointed out that withdrawal of the drug may not always prevent the development of agranulocytosis for the reason that changes in the white cell count of the peripheral blood do not in all probability occur until considerable damage has been done to the leucopoietic elements of the bone marrow. Practically all clinicians will agree that the continued administration of a drug known to cause agranulocytosis, once a reduction in the number of granular cells in the peripheral blood has begun to occur, is, to say the least of it, unwise; many would use a stronger term. In these circumstances a recent report by Norman Nixon, J. F. Eckert and K. B. Holmes[1] is certain to cause some comment by all who read it.

These observers report three cases of agranulocytosis which they hold were caused by sulphadiazine, and cure was effected, it is claimed, not by withdrawal of the drug, but by its continued administration in large doses. In the first case a cadet, aged twenty-six years, suffered from scarlet fever. The administration of sulphadiazine was started on the day the patient was admitted to hospital. From the seventh to the eleventh day of his stay in hospital the patient felt well and was afebrile; he then had what was regarded as a recrudescence of his scarlet fever. The number of white cells, as revealed in a leucocyte count, was falling, and the percentage of granular leucocytes was also falling. On the fifteenth day the patient again felt fairly well, but by this time the number of leucocytes was still further reduced. The administration of sulphadiazine was stopped on the fifteenth day, after a total of 58 grammes had been given. On the seventeenth day the leucocyte count reached 1,600 cells per cubic millimetre with 13% of granular cells. On the eighteenth day no granular cells could be found in many smears examined. On the nineteenth day the patient's general condition became worse, his temperature rose and it seemed that he would not recover. "Believing that death from overwhelming infection would probably occur, and surmising that possibly the agranulocytosis might be consequent to the effects of the hæmolytic streptococcus and not to the drug, it was reasoned that if infection could be held in abeyance long enough, the bone marrow would recover and the patient might be given a chance for survival." Other treatment, such as the use of liver extract, pentnucleotide, cevitamic acid and transfusions, was abandoned; sodium sulphadiazine was given intravenously twice daily in amounts sufficient to raise the blood level of the drug to twenty or twenty-five milligrammes per hundred cubic centimetres. Convalescent human scarlet fever serum was also given. Improvement began to occur after a day or two, and once granulocytes began to appear, recovery was rapid. On the twenty-eighth day there was no evidence of any blood dyscrasia.

It was after their experience with their second and third cases that Nixon, Eckert and Holmes changed their opinions and attributed the agranulocytosis in the first case to the sulphadiazine and not to the scarlet fever. The second and third patients suffered from virus pneumonia. The authors know of no instance in which agranulocytosis has resulted from virus pneumonia or other virus infection. On the other hand, sulphadiazine has been known to cause the condition, and as each of the three patients had received the drug for two or more weeks prior to the onset of the "bone marrow depression", Nixon, Eckert and Holmes are now "convinced" that the drug was responsible in all three cases. In the second and third cases, as in the first, it seemed obvious that the patients would die "unless the original infectious process and the later uncontrolled spread of infection were held in check during the period required for the temporary bone marrow depression to subside". It should be pointed out perhaps that the second and third patients received transfusions of serum from patients who were convalescent from virus pneumonia.

Though further details of the clinical histories of these patients cannot be recorded in this place, attention should be drawn to the fact that no record is made of any examination of the bone marrow by sternal puncture—this procedure might well have thrown light on these

[1] *The American Journal of the Medical Sciences*, December, 1943.

interesting cases. The first question that arises is whether the blood changes were due to the drug. If they were, it is not possible, in the absence of bone marrow examination, to determine how far the changes had progressed. Though it is possible that the convalescent serum given in each instance turned the scale in favour of the patients, the continued administration of sulphadiazine was none the less hazardous. Even though no granulocytes were to be found in the circulating blood, it could not be supposed that the drug had inflicted all the damage on the marrow that it was likely to inflict, that the damage to the leucopoietic elements of the bone marrow, once granulocytes disappeared from the circulation, would go no further. Clearly the damage in these three cases was not total or permanent, but it might well have been. Further conjecture is probably useless, but it must be concluded that, interesting and dramatic though the cases reported by Nixon, Eckert and Holmes undoubtedly are, they do not set a precedent that should be lightly followed. In the light of present knowledge clinicians will be very wary of doing as these authors have done. There may be some facts or factors about these three cases that have not come to light; there may be something fortuitous about them. On the other hand concealed in them may lie a germ of truth that will later on come to fruition. One is, of course, justified in wondering why the sulphadiazine was given at all.

"THE FEVER CURVE OF THERAPEUTIC FADS."

The man is fortunate who can look at himself and laugh, especially if the look follows something that has been said about him. He is still more fortunate if, having had his laugh, he makes good what has been lacking, corrects the mistake or curbs the excess of zeal that has given rise to the merriment. People of the British race, generally speaking, can be detached enough to look at themselves and laugh. They do not always, however, want to do anything about it. Doctors have just the same foibles, the same bursts of enthusiasm that other people have, but they have many that are peculiar to their calling—their peculiar geese often are depicted and treated as swans and their swans are apt to grow into birds of such a type that their progenitors would not recognize their offspring. Nowhere is this more noticeable than in the realm of therapeutics. Every medical practitioner has seen some drug or some form of treatment, newly introduced, boosted and used for almost every type of illness in which an excuse can be found for its exhibition. O. H. Perry Pepper, who is professor of medicine in the University of Pennsylvania School of Medicine, is a doctor who can look at himself and his brother doctors and laugh. He writes on what he calls "the fever curve of therapeutic fads", and his remarks should not be allowed to pass unnoticed.[1]

Our author with the merry name states that almost every new therapy seems to have to run the gamut of over-enthusiasm, disillusionment and neglect—such a course of events in the practice of medicine seems to be inherent and inevitable. It is possible to draw a graph of the progress of a therapeutic fad just as one plots the fever curve of an infectious disease. The picture of an infectious disease is the result of interactions between the causative agent and the host. A therapeutic fad does just the same kind of thing. "There must occur a period of invasion after the introduction of a new therapy during which occur prodromata in the form of reactions of interest displayed by the host. After a varying period the actual onset of the fad takes place with rapid spread in the use of the new medicament and a feverish interest in it. Soon the disease is at its height. The symptoms, in a classical instance, include at this stage an unwarranted enthusiasm for the curative effects of the new drug or treatment, an extension of its use for all sorts and kinds of ailments quite foreign to the claims of the original discoverer. Soon a delirious euphoria develops which sees the new therapy as a panacea for all ills. At this stage, the mind and reasoning power of the profession seem to be obtunded." Professor Perry Pepper points out that the fever of the fad is raised to even higher levels by the publication of reports of remarkable therapeutic results claimed for the method, some of the reports being hasty and ill-considered. To this should be added the careless reading and the uncritical interpretation of carefully prepared papers on a subject bearing on the "fad". But to continue with the symptomatology of this fever—neither the fever nor the "frenzy of the fad" ends by crisis. "The maintained height of the curve is broken by increasingly sharp falls with slowly returning consciousness and common sense." Failures are soon reported, and it is not realized that the successes occurred in properly selected cases and the failures in all others; indeed the criteria for proper selection may not be clear. "Unjustly, therefore, we turn against the method altogether, we forget its real value and discard it completely. The fever is 'broken' . . . and the disease is over." Convalescence is slow and final restoration to health is reached only when a proper and judicious use is made of what was once a frenzied fad.

The whole medical profession should be grateful to our Pennsylvanian professor of medicine for his courageous indictment. While on the one hand we can enjoy his method of attack, our enjoyment must be followed by some heart searching. His thrusts are sharp, and if we allow them to penetrate deeply enough we shall be the better for them. There is no need to refer to the fads described by him as occurring in successive ages of medicine. We have enough of our own day, and we see them all around us. Every doctor can probably say this, but what he needs to do is to find out whether he has any of his own. The faddist must try to remember that his addiction may do harm to a patient, though it is fortunately true that the human body can stand up to much therapy even as it can endure the toxins of many infections. Finally, the clinician is supposed to have a scientific training as a background to his therapeutic endeavours. If he neglects his science, his patients will suffer; and addiction to fads and the pursuit of science are mutually exclusive. If Professor Perry Pepper makes us remember at the right moment the trite saying that the *post hoc* is not necessarily *propter hoc* he will not have written in vain.

[1] *The American Journal of the Medical Sciences*, December, 1943.

BLEEDING GUMS AND GINGIVITIS IN NAVAL RATINGS.

An interesting investigation has been made by A. C. Macdonald into bleeding gums and gingivitis.[1] He examined 614 men from four ships in the Royal Navy. Of the total, 175 (25·5%) suffered from gingivitis; 134 suffered from mild and 41 from severe gingivitis. Mild cases were those in which clinical examination revealed red and swollen gums that bled readily on pressure or brushing. In severe cases the condition was of longer duration and was associated with fœtor, marginal ulceration, alveolar retraction and periodontal pocket formation; adenitis was seldom found. This was not the acute ulcerative gingivitis associated with Vincent's infection. Forty of the 614 men were edentulous. Of 411 men under thirty years of age, 17·7% were affected; of 183 over thirty, 37·7% were affected. Apparently vitamin deficiency was not a factor in causation. Vincent's bacilli and spirochætes were almost always present. Only local treatment was adopted, and consisted in scaling of the teeth with the application of 10% chromic acid solution followed shortly by peroxide of hydrogen. Treatment was effective, but there were some recurrences. Macdonald suggests that the routine scaling of the teeth is the most profitable form of prophylaxis. This is a practice that might be adopted with advantage for all civilians.

[1] *The Lancet*, December 4, 1943.

Abstracts from Medical Literature.

PATHOLOGY.

Sclerosing Hæmangiomata.

Robert E. Gross and S. Burt Wolbach (*The American Journal of Pathology*, July, 1943) state that certain hæmangiomata exhibit spontaneous, regressive changes which greatly alter their macroscopic and microscopic appearances. The initial step is usually dependent upon fibrosis which constricts capillary blood vessels, isolates segments of collapsed channels, and segregates groups of remaining endothelial cells. In about three-quarters of the instances there are accumulations of lipoid material or hæmosiderin or both. In occasional specimens these substances appear to come from hæmorrhages within the angioma, but this is not true in the majority of cases. The lipoid and pigment are presumably from, and extracted from, substances in the circulating blood. About one-quarter of the lesions show aggregations of endothelial cells forming foreign body giant cells. It is not surprising to find the type cell of hæmangiomata performing the dual role of blood vessel formation and phagocytosis, since these activities of endothelial cells are well recognized in the normal reticulo-endothelial system of the body. There is great variation in the prominence of fibrosis, vascular obliteration and phagocytic activity in sclerosing hæmangiomata. When connective tissue proliferation is advanced, the lesion is quite firm and greyish, and has the gross appearance of a fibroma or neurofibroma; microscopically, the original angiomatous nature of the specimen may be largely obscured. When phagocytosis of lipoid is an outstanding feature, the gross specimen may have a yellowish colour and the microscopic findings may be quite similar to some of the cutaneous xanthomata, but the condition has no relationship to the generalized lipoid disturbances. When pigment accumulates in high concentration, the gross lesion has a brownish colour and may be mistaken for a melanoma, but the microscopic appearance of the pigment and the positive reaction to iron stains differentiate it from melanin. The sclerosing process and the phagocytic properties may be exhibited by cavernous hæmangiomata and rarely by hæmangio-endotheliomata, but they are much more common in the capillary form of hæmangiomata. They may be found in hæmangiomata in many of the internal viscera, but they appear to be more frequent in those which are situated on the cutaneous surfaces of the body, particularly those of the trunk or the extremities.

Chondrosarcoma of Bone.

Chondrosarcoma is, according to Louis Lichtenstein and Henry Jaffe (*American Journal of Pathology*, July, 1943), to be regarded as a lesion distinct from osteogenic sarcoma of bone. The former develops out of a full-fledged cartilage, while the latter issues from more primitive tissue, arising in fact from bone-forming mesenchyme. Some chondrosarcomata do show large areas in which the intercellular matrix of the tumour cartilage has become heavily calcified or ossified, and in some osteogenic sarcomata cartilage in considerable amounts may be formed in the course of osteogenesis from the primitive mesenchyme. However, in a chondrosarcoma, in contrast to an osteogenic sarcoma, one never sees tumourous osteoid tissue and bone which is evolving out of a sarcomatous stroma directly, such as one always sees somewhere in an osteogenic sarcoma, no matter how much cartilage it contains. In comparison with osteogenic sarcoma, chondrosarcoma is less common, appears at a later age (on the average), runs a much slower course, and, especially if given radical surgical treatment at an early stage, has a much better prognosis, since the tumour has usually not yet metastasized at the time of initial surgical intervention. In the peripheral chondrosarcomata and those central chondrosarcomata which have clearly evolved from benign enchondromata, one finds, at least in the earlier stages of evolution of the lesion, heavy calcification or ossification of large parts of the intercellular matrix of the tumour cartilage. In other chondrosarcomata the relevant neoplastic tissue is likely to consist, in the main, of compacted islands of cartilage with hyaline matrix, though if the chondrosarcoma is very bulky, areas may also be seen in which the cartilage is softer and myxomatous, and perhaps even necrotic. With regard to the differential diagnosis between benign chondroma and chondrosarcoma, the authors hold that a cartilage tumour should no longer be regarded as benign if, when viable and not heavily calcified areas are examined, it shows even in scattered fields: (*a*) many cells with plump nuclei; (*b*) more than an occasional cell with two such nuclei, and especially (*c*) any giant cartilage cells with large single or multiple nuclei or with clumps of chromatin. They feel that, by the observance of these criteria, the prevalent tendency to "underdiagnosis" of chondrosarcoma in an early stage of malignancy can be overcome.

Cystosarcoma Phylloides.

W. G. Cooper, junior, and L. V. Ackerman (*Surgery, Gynecology and Obstetrics*, September, 1943) report three cases of *cystosarcoma phylloides* of the female breast, one of which metastasized to the axillary lymph nodes. *Cystosarcoma phylloides* is the most common of scores of names used for this tumour. The authors therefore retain it with the following definition: it is a tumour arising from the connective tissue of a preexisting mammary adeno-fibroma. The clinical history usually obtained is that the patient in her fourth or fifth decade has been aware of a quiescent, small, hard breast nodule for many years with later growth to a huge size without associated constitutional symptoms. On examination, the breast is found tremendously enlarged and characteristically ovoid in shape. The overlying skin and nipple are not invaded, although local extension may have taken place to the adjacent structures. Regional lymphadenopathy is not evident unless infection is superimposed. Grossly, the tumour is found to contain frond-like pleats resembling a head of cauliflower, from which appearance it takes its name. Microscopically, the unique finding is the increase in the fibrous elements. The associated metaplastic changes, areas of ectopic cartilage or bone, and acinar proliferation which sometimes occur are not of definitive value. "Atypical carcinoma", lymphosarcoma and diffuse fibrosarcoma of the breast must be rigidly excluded from this group. These patients usually do well when treated by wide local excision, and the prognosis in general is good.

Gynandroblastoma of the Ovary.

Emmett A. Mechler and William Black (*American Journal of Pathology*, July, 1943) state that ovarian tumours exist in which there is alteration of the secondary sexual characters of the bearer in the male direction, presumably as a result of the androgenic hormone liberated by the tumour; at the same time there is a continuation of cyclic menstrual bleeding, with indications of hyperœstrinism. Such cases differ from granulosa cell tumours both biologically and histologically. Robert Meyer first applied the term gynandroblastoma to them. There is no constant cell pattern in these cases, and they appear to represent combinations of granulosa cell tumours and arrhenoblastomata. Interstitial cells carrying lipoids, resembling the testicular Leydig cells, are common to both gynandroblastomata and arrhenoblastomata. There is presumptive evidence that these cells are the source of the androgenic hormone. The authors review from the literature twenty cases which may belong to this group, and they add a case of their own. A woman, aged twenty-four years, had menstruated regularly until four months before her admission to hospital. Then the intervals became shorter, and for the last ten days there had been continuous vaginal hæmorrhage. In spite of these and other signs of undiminished œstrogenic activity, there had been over the last five years a gradual development of general hirsutism, beard, male voice and enlarged clitoris. A tumour of the left ovary was found and removed. Microscopically, it showed structures which were consistent with a granulosa cell tumour in some parts, with an arrhenoblastoma in other parts. After operation, menstruation became regular again, and has remained so for three years. The features of virilism have become less conspicuous, but no complete regression has occurred.

Relation Between Hepatic and Plasma Concentrations of Vitamin A in Human Beings.

H. Popper, F. Steigmann, K. Meyer and S. Zevin (*Archives of Internal Medicine*, October, 1943) present a study performed by chemical methods and fluorescence microscopy which is concerned with the comparison between the plasma vitamin *A* level and the hepatic vitamin *A* concentration in human subjects under normal and under pathological conditions. The plasma vitamin *A* level shows great variations brought on by varied nutritional intake and by disease. It is moderately reduced in carcinoma of the gastro-intestinal tract and greatly reduced in hepatic damage and in jaundice. Anæsthesia has little, if any, influence on the plasma vitamin *A* level. Comparison between the plasma level and the vitamin *A* concentration

in biopsy specimens of liver over a wide range of the plasma vitamin *A* level (patients with jaundice, normal patients and patients receiving huge feedings of vitamin *A*) shows a parallelism between plasma and liver vitamin *A* concentrations only if the average of a fairly great number of cases is taken. If individual cases are considered no relation between these two factors exists. The plasma vitamin *A* level is thus no index of the liver stores of the vitamin, except that a high plasma level indicates normal or high stores. In contrast, low plasma levels may be associated with high or low liver contents of vitamin *A*. The fluorescence microscopic picture shows great variations of the vitamin *A* fluorescence pattern. These variations are extremely marked in pathological conditions of the liver and offer a possibility for the recognition of hepatic damage morphologically before it is revealed by routine histological examination. The disturbance of the pattern is pronounced when there is great discrepancy between plasma and liver concentrations of vitamin *A*. Since apparently the plasma level is regulated by the liver, these pathological changes in distribution of vitamin *A* explain disturbances in the release of the vitamin and the consequent discrepancy. In hepatic disease and in jaundice this disturbed release seems to be common. In general, the plasma level seems a better index for the functional status with regard to vitamin *A* than do the liver stores. Intake of huge doses of vitamin *A* raises the concentration in the plasma much more than in the liver. This increase is less pronounced in jaundice. The amount of vitamin *A* fluorescence as seen under the microscope and the chemical assay for vitamin *A* (the method of which is discussed) run parallel in the human liver. If high amounts of vitamin *A* are present, they are chiefly found in Kupffer cells.

MORPHOLOGY.

Epithelial Repair in the Cornea.

L. B. AREY AND W. M. COVODE (*The Anatomical Record*, May, 1943) state that the events responsible for the epithelial healing of wounds have been identified variously in the past. Cell division, cell migration, connective tissue metaplasia, and different combinations of these factors, have all had their proponents at one time or another. But the principal question has centred on whether epithelial migration or cell division is primary. The present investigation shows that the repair of wounds in the corneal epithelium of the white rat is accomplished by cell migration which fills the defect promptly. During this primary period of reepithelization the mitotic rate falls far below normal in the neighbouring epithelial territory, while cell division is lacking, or virtually lacking, in the area actually being reclothed. Although the new epithelial cover was found to be complete within twelve hours, it was not until ninety-six hours that the reduced mitotic rate increased again markedly. Even then it was in the more remote epithelium, rather than in the wound site proper, that mitoses occurred in any abundance. The greatest mitotic activity was shown at 120 hours. During the six days under observation the frequency of cell division, as calculated on the basis of unit epithelial area, never exceeded the normal rate. Epithelial area, rather than volume, is the more significant basis for comparison. The occurrence of mitosis in the normal cornea of the white rat is about 750 per square millimetre of epithelium, and about 16,750 per cubic millimetre. A young, sexually immature rat showed no significant difference in mitotic frequency from two older animals. Cell division is relatively frequent in corneal epithelium —about four times the rate in the œsophagus. It is possible that the cornea merely retains the vigorous proliferative capacity which is enjoyed by young tissues in general, but which is greatly reduced at maturity in most of these tissues.

Testicular Descent.

N. R. WYNDHAM (*Journal of Anatomy*, January, 1943) gives an account of the development of the structures associated with descent of the testis in embryos ranging in size from 12 millimetres to 27 centimetres C.R. length. The present study does not support the view that the inguinal canal grows up to envelop the testis, and as very little connective tissue was found in the gubernaculum, there is little support for the view that the testis is pulled down by scar contraction of the gubernacular connective tissue. Descent seems to take only a short time, and just before it occurs, the testis is three centimetres from its final position. The author expresses the opinion that the intense vascularity, the increase in size of the testis prior to its descent and the fact that the striated muscle fibres of the gubernaculum are in the plane of the inguinal canal must all have some significance. He also emphasizes the fact that the testis has a mesentery and that it is not a retroperitoneal organ like the kidney, free to slide down the abdominal wall behind the peritoneum.

Bipolar Cells in Hypoglossal Nerve.

A. A. PEARSON (*The Anatomical Record*, April, 1943) discusses the problems concerning a sensory component in the hypoglossal nerve. The majority of authors recognize the probability of a sensory component in the hypoglossal, but there has been much speculation concerning the possible course and location of the sensory neurones. The occurrence of dorsal roots with ganglia, such as the Froriep's ganglia in certain ungulata, is considered extremely rare in man. In the present communication, the author reports the discovery of bipolar neurones in the intramedullary course of the hypoglossal nerve in the region of the hilus of the inferior olivary nucleus in human fœtuses of four, five and six months. The character of these neurones suggests that they are sensory in function.

Hæmopoiesis in Chorionic Villi.

G. B. WISLOCKI (*The Anatomical Record*, April, 1943) confirms the discovery of J. P. Hill that erythropoiesis occurs in certain dilated blood vessels of the chorionic villi of the placenta of the marmoset and other platyrrhine monkeys. Erythropoiesis makes its appearance in the placenta of platyrrhine monkeys some time after the establishment of the circulation, but disappears completely toward the end of pregnancy. Study of the liver and spleen of two marmoset fœtuses suggests that placental erythropoiesis acts as a substitute in platyrrhine monkeys in large measure, during the first half of gestation, for these organs which in other animals are the usual and dominant sites of blood formation during fœtal life.

Aging of Human Skin.

R. EVANS, E. V. COWDRY AND P. E. NIELSON (*The Anatomical Record*, August, 1943) report results of an investigation to clarify the rather nebulous concept in senile atrophy of the skin. Stained sections of skin show striking differences between young and old, but the authors conclude that most of the difference is the result of greater shrinkage of young connective tissue, for it was found that removal of the dermis abolished much of the difference.

Gross Variations in Ileo-Caecal Valve.

R. E. BUIRGE (*The Anatomical Record*, July, 1943) states that the classical type of ileo-caecal valve was observed in 54% of 500 post-mortem specimens. Anatomical variations of the ileo-caecal junction are classified according to observed deficiencies of the *frenula valvuli coli*. It is evident that sufficiency or insufficiency of the ileo-caecal valve or sphincter may depend on its structure. A case is reported in which incompetency of the ileo-caecal junction was associated with the author's Type III caecum and a Type II (A) valve.

Lymphatics of Tunica Vaginalis.

L. ALLEN (*The Anatomical Record*, April, 1943) states that the parietal *tunica vaginalis* of man possesses a lymphatic plexus comparable to the subserous plexuses of the diaphragm and intercostal pleura. This plexus drains into the same regional lymph nodes as the lymphatics of the parenchyma of the testis. Development of a lymphatic plexus in the parietal *tunica vaginalis*, like obliteration of the proximal portion of the *processus vaginalis*, is peculiar to man. An hypothesis is advanced in this connexion in regard to the most frequent congenital defects of *processus vaginalis*.

Factors Influencing Development of Scrotum.

H. SELYE (*The Anatomical Record*, April, 1943) states, as a result of experiments, that both hormonal and mechanical factors play a role in scrotal development. Testosterone in itself does not induce full scrotal development in immature rats, nor does it completely inhibit scrotal involution in adults which have been deprived of their gonads unless the scrotum is mechanically distended. Distension by the epididymis or an "artificial glass testis" can sensitize the scrotum to the action of testosterone. Without this mechanical stimulus the scrota of castrate rats remain subnormal even though the animals receive testosterone in doses sufficient to develop the seminal vesicles and prostates far beyond their normal size.

XUM

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on October 28, 1943, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. K. S. M. BROWN, the President, in the chair.

Malaria.

LIEUTENANT-COLONEL E. T. BRENNAN, D.S.O., M.C., read a paper entitled "The Treatment of Malaria" (see page 189).

In answer to a question of a member of the United States Army Medical Corps as to how long after the intramuscular injection of quinine one might expect to see an improvement in the condition of a patient who was suffering from the cerebral form of malaria, Lieutenant-Colonel Brennan said that the effect of an intramuscular injection of quinine was very quick, if it was given early enough; otherwise improvement was somewhat delayed. As a rule the patient responded within a few hours, except in very severe cases, such as those in which coma lasted for two or three days, in which cases intravenous injections would be given. In those circumstances it was necessary to keep pushing quinine in. In answer to questions whether sulphapyridine acted on all types of malaria and whether penicillin had been tried in malaria, Colonel Brennan replied that he did not know.

DR. W. L. CALOV thanked Colonel Brennan for his paper, which was of particular value, coming from one who had had very many years' experience in a malarious country, and from one who had recently returned from that country. Dr. Calov was disappointed that few of the medical personnel of the services were present. Colonel Brennan had mentioned "Atebrin" in malignant tertian malaria, stating that in Europe it had been found to be more efficacious than quinine in this type of malaria, but not in New Guinea, where the condition was brought better under control with quinine. The same statement applied to the whole of the south-west Pacific. Dr. Calov had had a striking example some years previously, when "Atebrin" first came into use. A patient from New Guinea consulted him, suffering from severe subtertian malaria. Dr. Calov treated him with "Atebrin" in the approved doses; but in four days the temperature was still much elevated, and many subtertian parasites were to be found in the blood. In great trepidation, because he did not then know the effect of a combination of the two drugs, Dr. Calov gave the patient fifteen grains of quinine intramuscularly, and the next morning the patient was able to sit up and take breakfast. Dr. Calov went on to say that the staining effect of "Atebrin" was interesting: he had formed the impression that if no staining occurred, or if the urine was not stained, "Atebrin" was not being absorbed. He wondered whether Colonel Brennan could shed any light on the subject. American "Atebrin", as far as he knew, was the same as the German "Atebrin": it was supposed to have the same chemical formula. But patients had declared to him that they could not recover under treatment with the American drug, and that if they could only obtain some of the German product, they would be right. Dr. Calov himself believed that the theory was without foundation. Dr. Calov then referred to the idea that if a patient suffering from benign tertian malaria and having rigors was deprived of all the blankets and covers he wished to put on, he would not sweat. Dr. Calov said that he himself had had rigors, and if he was denied the use of blankets, overcoats, sheepskin rugs and anything else he wished to put on his bed, he would be most annoyed. The rigor was the most uncomfortable stage, not the sweating stage. By the time the patient was sweating heavily he was feeling comfortable and was able to turn over and go to sleep. Dr. Calov said that he would not deprive his patients of all the covers they wanted. Colonel Brennan had mentioned gastro-intestinal forms of subtertian malaria. It was possible that he, as well as Dr. Calov, had seen an unfortunate abdomen submitted to surgical operation for some acute abdominal disease, when the patient was really suffering from malaria. Dr. Calov remembered one such patient whom the surgeon thought to be suffering from a ruptured gastric ulcer. The abdomen was opened at the epigastrium. No particular abnormality was found, so the surgeon opened the abdomen a little lower down and removed the appendix. The patient recovered, because later on he was given quinine. Dr. Calov had seen patients suffering from malaria who presented a typical clinical picture of appendicitis; possibly one might err in the opposite direction. Dr. Calov went on to say that the treatment of relapses of benign tertian malaria was assisted by "ringing the changes" between "Atebrin" and quinine. That procedure was recommended by the Health Committee of the League of Nations. One relapse would be treated with quinine, the next with "Atebrin", and so on. It was possible that the infection was eliminated more rapidly in that way. Many of the so-called relapses of malaria of which people complained in Australia were not really relapses of malaria at all. People consulted Dr. Calov years after they had left malarious countries, complaining that they had just had a bad bout of fever. Dr. Calov said that once a person had had malaria, it was likely that he would ascribe any subsequent febrile illness to malaria. Dr. Calov had found that many such people were suffering from pyelitis, and he wondered whether splenic enlargement and some disturbance of the anatomical structure of the pelvis of the kidney and ureter might not have something to do with it. Dr. Calov was glad that Colonel Brennan had stressed the importance of intramuscular injections of quinine. He said that he had never seen any disasters follow their use, apart from the ghastly pictures in text-books. He thought that, in the cases from which those pictures were taken, the injection had possibly not been given by a person with a knowledge of asepsis.

DR. E. H. M. STEPHEN asked Lieutenant-Colonel Brennan whether the onset of attacks of malaria was often accompanied by convulsions in early childhood. He was prompted to ask this question by having had a patient in whose case this had happened. He did not remember having seen this pointed out in text-books on the subject.

DR. B. A. SINCLAIR said that after Colonel Brennan's excellent and comprehensive paper there was little one could say except to give a few personal experiences. With regard to quinine the optimum daily dose for an adult would appear to be thirty grains, for he had seen patients who had taken twenty grains daily for a week without complete relief of symptoms, but whose malaria cleared up rapidly with a daily dosage of thirty grains. Dr. Sinclair had always given it in divided doses of ten grains with each meal, starting treatment at the first meal hour irrespective of whether the temperature was rising or falling and continuing the thirty grains daily only so long as any symptom or temperature remained, then instructing the patient to continue with either five grains or ten grains as suppressive treatment. Many years ago he gave up the scheme of progressive reduction of daily dosage, for example, thirty grains daily for one week, then twenty grains daily for a week, then ten grains daily for a week, because he found that patients would rarely be bothered with it when discharged from hospital, and also, that those who treated themselves at home with merely enough quinine to abolish symptoms and then carried on with suppressive dosages did as well as those who took the more massive course. For women, generally twenty-five grains daily were sufficient. It did no harm in pregnancy, but might cause floodings at the menses. The patients in this type of case and those with impaired hearing would be grateful to the doctor if he avoided ordering them to take quinine.

With reference to "Atebrin", Dr. Sinclair said that he was interested to note that in Stitt's 1943 edition of "Tropical Diseases" he quoted: "Peter has shown that foods with large amounts of cellulose in them are apt to absorb Atebrin." Dr. Sinclair had recovered from one motion two "Atebrin" tablets showing no sign whatever of their journey through the intestinal tract; since then, "Atebrin" tablets in hospital practice had always been broken in half before being given to the patient. It was very bitter, much like quinine. Gunther, of Bulolo, had a hospital patient develop rigors on the fifth day of an "Atebrin" course of one tablet taken three times a day. He told Dr. Sinclair that there were numerous parasites in the film on that day. Dr. Sinclair had also had a similar experience. In view of the statement that foods with much cellulose might absorb "Atebrin", and also the finding of unaltered tablets in excreta, it became easier to understand those failures with "Atebrin" that were met from time to time.

For about the last five years it had been Dr. Sinclair's practice to give six 0·1 gramme tablets of "Atebrin" daily for only so long as temperature or symptoms existed, for example, two to three days, then the patient was advised to take suppressive doses of "Atebrin" of two tablets on each of two successive days. A lot might be learnt by asking the patients which suited them the better, quinine or "Atebrin", and how much of either they found necessary to relieve them of symptoms, for this was a varying factor with most people. In the simple type of malaria confinement to bed was

necessary only so long as there was a rise in temperature, and this varied from half a day to three days. Treatment should be of the simplest, apart from anti-malarial drugs. Unnecessary aperients were to be avoided, and to insist on the taking of nauseating alkalis only made the patient uncomfortable. Diet was of little importance, and no purpose was served by weakening the patient by restricted diet if he felt like taking a full diet. Quinine might cause insomnia or nightmare, and a bromide or other sedative at night was always welcome, if the patients declared quinine so affected them. After cessation of fever or symptoms a period of forty-eight hours for convalescence was sufficient before the patients returned to work. Dr. Sinclair thought that the average time away from work in these cases in New Guinea would be four days, irrespective of whether the patient was a manual or a brain worker. Gastric malaria was very common, and a very distressing ailment, for these patients would vomit everything taken by mouth. However, it was generally very amenable to treatment. As soon as the condition was diagnosed a hypodermic injection of a quarter of a grain of morphine and an intramuscular injection of a gramme of quinine should be given, and within two hours vomiting generally ceased. Time should not be wasted in making the patient sicker with all the various gastric sedatives; they had a very nasty taste on the return voyage. The treatment mentioned worked like a charm.

Blackwater fever, so far as Dr. Sinclair could recollect, was the only malarial cause of death among Europeans that he saw in fourteen years in New Guinea. It was said that quinine, "Atebrin" or "Plasmoquine" might precipitate an attack. Dr. Sinclair had never seen a case originate in hospital practice in spite of these drugs being given in full therapeutic doses on the patient's admission. It was a very serious manifestation of malaria. Consideration had to be given to the injection of either "Atebrin" or quinine if the condition persisted and parasites remained in the blood film. These measures were heroic, but sometimes life-saving. Once infection with the specific parasite occurred, the disease might occur both in or outside the malarious areas. Vomiting was frequent, and intravenous infusions, blood transfusions *et cetera* were of major importance. It was also extraordinary how the urine could clear rapidly, and as rapidly become almost black again. Dr. Sinclair had once had a patient who in spite of two blood transfusions was showing no improvement and whose urine cleared like magic when he was given an intravenous infusion of 2% saline and 10% glucose solution and he progressed rapidly to health. Aperients should be avoided lest they provoked a diarrhœa, and alkali mixtures were best left alone unless the patient was free of any nausea. Cerebral sedatives were highly important to allay anxiety and restlessness.

Cerebral malaria was not common in Dr. Sinclair's experience and usually responded well to injections of quinine, except the severely comatose types. In the latter immediate intravenous injections had to be given and repeated at eight hour intervals whilst coma persisted. Lumbar puncture was helpful; also, of course, attention had to be given to the bladder.

The question of whether quinine should be injected intramuscularly seemed to be a controversial one. Every textbook gave warnings of dire results such as abscesses, gangrene and so on. In Dr. Sinclair's experience there had been no evidence to support this condemnation of a most excellent form of therapy. He must have given between 500 and 1,000 intramuscular injections of quinine to persons of all ages in all physical conditions, and practically all to Europeans. He had never seen any dire result nor heard of any in the work of any colleague in New Guinea. Dr. Sinclair's wife would have had between 70 and 100 and still preferred it as the method of treatment. A matron of one of the New Guinea hospitals had told Dr. Sinclair that she had had between 30 and 40 injections in her time, and she was a strong advocate for the method. Some text-books ascribed abscesses to giving a second injection into the same buttock. Some said it was dangerous to inject onto the bone. Dr. Sinclair found little evidence to support these statements. One could inject quinine too shallowly, but never too deeply. Injection into the fat of the buttocks might cause a painful nodule, but this was not an intramuscular injection. Dr. Sinclair always gave three injections into the same buttock, spacing the points of entry in a triangular form, each point being about half an inch from the others. He always injected one gramme (fifteen and a half grains) each time. An injection was given on each succeeding day and no other antimalarial drug was given orally. In thin people the needle was pushed down till bone was felt, then withdrawn a quarter of an inch, and then the injection was made. No collodium or sticking plaster was used to cover the needle hole. It was very common with the first injection, just as it was started, for the patient to feel a cramp-like pain down the back of the leg, but this was momentary and of no consequence. Maximum absorption appeared to take about two hours, judging by clinical effects. He had seen one abscess from an intramuscular injection of "Atebrin", but healing was rapid once the pus was let out. Patients could receive intramuscular quinine injections and might carry on fully with their occupations.

Colonel Brennan's statement about developing immunity to a particular strain was very interesting. When coming away from New Guinea, a party including Dr. Sinclair had to sleep nightly for three weeks in heavily endemic malarious villages. In order to keep fit, each man was given ten grains of quinine every day, and only one man developed any malaria during the trek. However, most of those in the party must have been freshly infected, for in general those whom Dr. Sinclair had since contacted had had more malarial relapses in the first twelve months than they would have over a period of years in New Guinea. Dr. Sinclair himself had had ten relapses in that period, but would be incapacitated only for about five hours of the day of the relapse. For the first five attacks he took quinine thirty grains daily for three days, then "Atebrin" 0·1 gramme, six such tablets, daily for seven days. For one attack he took quinine thirty grains daily for ten days, but these courses were of no avail. He was decided now more than ever that the quinine and "Atebrin" taken after temperature and symptoms had subsided were waste of drugs, and he came back to the old scheme of taking only enough of either to knock the attack. The last two relapses had been satisfactorily treated with only ten grains of quinine and nothing else. Should he have another relapse, Dr. Sinclair proposed to allow immunity to control it, and not to take any quinine or "Atebrin". If a patient was suspected of suffering from malaria, a blood film should be taken, and treatment started immediately without waiting for the result of the blood film. A "negative" report did not necessarily mean no malaria. In conclusion, Dr. Sinclair said that it had to be borne in mind that the remarks he had made were in reference only to malaria as seen in Europeans in New Guinea during peace time, and any reference to quinine meant quinine bi-hydrochloride.

Flight-Lieutenant F. H. Read (Royal Australian Air Force) referred to the occurrence of minor malarial attacks in people who had left malarious districts six months or more previously, and he wondered what kind of treatment should be adopted. He had recently encountered a case in point. As Colonel Brennan had pointed out, it seemed that it was possible to rely on the history given by the patient. A sergeant, who had served in New Guinea, about seven months prior to the meeting had had malaria. He had had no attacks since he returned to Australia, and had been in good health until about three weeks prior to the meeting, when he suddenly suffered from giddiness, nausea and severe headache and backache. The patient was quite sure in his own mind that he was suffering from malaria, because the symptoms were similar to those he had had in New Guinea. Flight-Lieutenant Read examined a blood film and had a blood count made. No parasites were to be seen. The erythrocytes numbered 4,100,000 and the leucocytes 11,000 per cubic millimetre, and the hæmoglobin value was less than normal. The patient's temperature never rose above 99° F., but he was given a course of treatment with quinine, "Atebrin" and "Plasmoquine". Soon after the course had begun he started to get better. The point of interest was that the patient was quite sure that he was suffering from malaria, and although it could not be proved, the attack cleared up under antimalarial treatment. Flight-Lieutenant Read said that possibly a number of such minor attacks would be encountered. Was it advisable to carry on with a course of suppressive treatment? And if so, for how long?

Lieutenant-Colonel Brennan, in reply to the question concerning the use of quinine for surgical patients, said that he had always given full quinine therapy while the patient was having surgical treatment, if there was a history of recent malaria, or if parasites were present in the blood. Colonel Brennan also gave full quinine therapy in obstetric cases, and to pregnant women who had had recent malaria before they were confined. It was much better and safer to give such women antimalarial treatment; abortions were more likely to occur if malaria was untreated than if they were given quinine. Colonel Brennan did not think that quinine interfered with any surgical treatment. In reply to Dr. Stephen, Colonel Brennan said that convulsions were common in children suffering from malaria, especialy benign

tertian malaria. If the child's temperature rose above 104° F., convulsions almost always occurred. In reply to Flight-Lieutenant Read, Colonel Brennan said that the patient mentioned was obviously suffering from chronic benign tertian malaria, in which parasites were often not found except during the patient's first shivering attack, when his temperature was rising.

CAPTAIN H. ROOT, of the United States Army Medical Corps, thanked Lieutenant-Colonel Brennan for his interesting paper. He said that he and his colleagues who were present were not prepared to discuss the paper; they had come to the meeting to learn.

DR. K. S. M. BROWN, from the chair, said that the address given by Colonel Brennan had been interesting and instructive. Dr. Brown regretted that so few civilian practitioners were present to hear it, because most of them encountered once or twice during the year a case of malaria. It would be necessary for them all to know more about the diagnosis and treatment of malaria in the future. On behalf of the New South Wales Branch, Dr. Brown thanked Colonel Brennan and those who had taken part in the discussion.

MEDICO-POLITICAL.

A MEETING of the Victorian Branch of the British Medical Association was held at the Royal Australasian College of Surgeons, Spring Street, Melbourne, on February 9, 1944, DR. D. ROSEBY, the President, in the chair.

Statement of Receipts and Expenditure.

Dr. C. H. Mollison, the Honorary Treasurer, presented the statement of receipts and expenditure of the Victorian Branch of the British Medical Association and of the Medical Society of Victoria for the year ended December 31, 1943. In presenting the statement Dr. Mollison spoke in the following terms.

I have pleasure in presenting the statement of receipts and expenditure of the British Medical Association and the Medical Society of Victoria for the year ended December 31, 1943.

The credit balance at December 31 was £1,305 9s. 6d., a decrease of £315 13s. 10d. compared with the previous year. The principal reasons for the decrease are set out hereunder. The amount received from subscriptions during 1943 was £7,661 14s. 8d., as against £6,851 5s. 4d. in 1942, an increase of £810 9s. 4d. Subscriptions for 1943, however, include £863 7s. 9d. for the Medical Society of Victoria Organization Fund, which was not in existence during 1942. The comparable figures for two years are therefore £6,798 6s. 11d. for 1943 and £6,851 5s. 4d. for 1942, a decrease of £52 18s. 5d. in 1943.

Whilst the number of subscriptions paid in 1943 was greater than in 1942, the increase was mainly in the sections paying the lower rates of subscription, thus reducing the average amount of subscriptions paid per member exclusive of that portion allocated to the Organization Fund.

The reasons for this reduction are:

1. The number of members who were entitled to the reduced subscription of £1 11s. 6d. applicable to members of the forces increased from 352 at December 31, 1942, to 419 at December 31, 1943.

2. Some members who joined the forces in 1942 had paid the full subscriptions prior to enlistment and had sufficient credit to cover the 1943 subscriptions, whilst some who had joined prior to 1942 were still in credit for the whole or portion of the 1943 subscription.

3. The increase in junior members, some of whom were liable for the subscription for six months only.

4. Arrears of subscriptions paid in 1943 were less than in 1942.

Expenditure increased by £530 13s. 1d.

Items in which there were increases were: bonus to Medical Secretary £200; payments to London £56, Australasian Medical Publishing Company, Limited (THE MEDICAL JOURNAL OF AUSTRALIA) £48—due in both cases to the increased number of subscriptions paid; rebates to subdivisions £11; salaries £113—due to increases to two typistes and stenographers and the caretaker; stationery £119; postages £77; printing £4; heating and lighting £13; telephones £8; and library (mainly on binding) £8.

The increase in the expenditure on postages is due to the increased number of Branch meetings and the increased activities of the Branch.

The same reasons apply with regard to the expenditure on stationery. A further reason for the increased expenditure on stationery was that during the year, in view of the prospect of short supply, it was considered desirable to purchase in advance of normal requirements, so that at present there is a fair quantity of stationery on hand which, under normal conditions, should be reflected in a reduction in expenditure under this head in 1944.

The increase in heating and lighting was due to the necessity, owing to weather conditions, to continue the use of the electric radiators to a much later date than usual.

On the other hand, there were decreases in expenditure under the following heads.

Federal Council capitation fees, £25—due to the fact that the 1942 expenditure included £40 for arrears for the year 1941.

Rates, taxes and insurance, £44. The expenditure for 1942 included contributions to war damage insurance for 1942 and 1943 on contents of buildings and for 1942 on the buildings, whereas the 1943 expenditure was for contribution for the buildings only.

Repairs. The only expenditure under this head in 1943 was £1 2s. 9d. for repairs to radiators. Repairs to the building were carried out during the year, payment for which was made in January last.

Interest on debentures, £14. Debentures of the face value of £350 were transferred to the British Medical Insurance Company at a reduction in interest from 5% to 1% *per annum*.

I have placed the expenditure under the different items for the respective years in juxtaposition, so that if any member desires details, I am in a position to furnish them.

	1942 £	1943 £
B.M.A.—London	1,975 6 3	2,031 14 3
Rebates	22 14 0	34 2 6
Federal Council capitation fees	502 4 0	477 12 0
Salaries	1,981 4 10	2,094 7 3
Mr. Crouch	300 0 0	300 0 0
Postages	138 10 3	215 12 1
Stationery	66 19 1	186 8 3
Printing	13 14 10	17 18 4
Sundries	95 3 9	84 6 6
Audit	15 15 0	15 15 0
Bank charges	3 18 1	4 13 4
Telephones	64 8 9	72 12 3
Travelling expenses	3 7 8	3 1 10
THE MEDICAL JOURNAL OF AUSTRALIA	1,362 0 0	1,410 10 8
Library books *et cetera*	128 16 5	138 1 3
Rates, taxes and insurance	103 13 8	59 16 3
Legal expenses	1 1 0	—
Lighting and heating	44 13 9	57 14 1
Entertainment	11 7 0	11 15 6
Donations	1 1 0	2 2 0
Repairs and alterations	38 13 0	1 2 9
Debenture interest	222 5 0	208 5 0
Bonus to Medical Secretary	—	200 0 0
	£7,096 17 4	£7,627 10 5

Members may rest assured that strict vigilance is being kept over all expenditure.

The association is again greatly indebted to the insurance company. In addition to providing £480 towards the Medical Secretary's salary, its action in voluntarily agreeing to a reduction in the interest rate of its debenture holding from 5% to 1% represents a substantial saving to the society. The company now holds debentures of the society to the extent of £2,950 on which interest at the rate of 1% is paid. This represents a saving of £118 per annum in interest charges. The company has also purchased books for the library to the value of £101 10s. 9d. I trust members will show their appreciation for the assistance given by the insurance company by placing as much of their insurance with it as they can possibly arrange. Members should bear in mind that this company is theirs and that its profits will accrue to their good. The company is an asset which members should take particular care to conserve and enhance.

The amount contributed to the Medical Benevolent Fund, £224 15s. 9d., is £23 19s. 7d. more than the previous year. I would particularly draw members' attention to this fund. It is one that should need no recommendation from me. Its sole purpose is to relieve distressed members. Unfortunately its activities have to be restricted to the degree to which members contribute to the fund.

I have pleasure in submitting for adoption the statement of receipts and expenditure.

The financial statement, which was adopted, is published herewith.

BRITISH MEDICAL ASSOCIATION (VICTORIAN BRANCH) AND MEDICAL SOCIETY OF VICTORIA.

Combined Statement of Receipts and Expenditure for Year ended December 31, 1943.

RECEIPTS.	£ s. d.	£ s. d.
To Balance at December 31, 1942—		
National Bank of Australasia Ltd.—		
Medical Society of Victoria State Savings Bank of Victoria	829 12 9	
Commonwealth Loans	500 0 0	
Cash in Hand	12 0 0	
	1,841 12 9	
Less National Bank of Australasia Ltd. Overdraft, B.M.A.	220 9 5	
		1,621 3 4
„ Subscriptions		7,661 14 8
„ Medical Benevolent Fund		224 15 9
„ British Medical Insurance Co.—Grant		480 0 0
„ Rent of Hall		3 16 0
„ Sale of Journals		5 2 0
„ Interest on Investments		24 11 8
		£10,021 3 5

EXPENDITURE.	£ s. d.	£ s. d.
By British Medical Association, London		2,031 14 3
„ THE MEDICAL JOURNAL OF AUSTRALIA		1,410 10 0
„ Medical Benevolent Fund		224 15 9
„ Federal Council of the B.M.A.—Capitation Fees		477 12 0
„ Rebates to Subdivisions		34 2 6
„ Medical Society of Victoria—Organization Fund		863 7 9
„ Library Books and Journals *et cetera*	239 12 0	
Less Contribution from B.M.I. Co.	101 10 9	
		138 1 3
„ Salaries and Bonus—		
Medical Secretary	1,200 0 0	
Office and Library *et cetera*	1,094 7 3	
		2,294 7 3
„ Pension—Mr. C. S. Crouch		300 0 0
„ Debenture Interest		208 5 0
„ Audit Fees		15 15 0
„ Postages		215 12 1
„ Printing and Stationery		204 6 7
„ Sundry Expenses		84 6 6
„ Bank Charges and Exchange		4 13 4
„ Telephones		72 12 3
„ Lighting and Heating		57 14 1
„ Rates, Taxes and Insurance		59 16 3
„ Repairs		1 2 9
„ Travelling Expenses		3 1 10
„ Entertainment		11 15 6
„ Donations		2 2 0
„ Balance at December 31, 1943—		
National Bank of Australasia Ltd.	1,159 13 5	
Medical Society of Victoria—		
State Savings Bank of Victoria	508 6 8	
Commonwealth Loans	500 0 0	
Cash in Hand	12 0 0	
	2,180 0 1	
Less National Bank of Australasia Ltd. overdraft, British Medical Association	874 10 7	
		1,305 9 6
		£10,021 3 5

(Signed) C. H. DICKSON, Secretary. (Signed) CRAWFORD H. MOLLISON, Hon. Treasurer.

Compared with the Books and Accounts of the British Medical Association, Victorian Branch, and the Medical Society of Victoria and found to be in accordance therewith.

Melbourne,
January 21, 1944.

(Signed) J. V. M. WOOD AND CO.,
Chartered Accountants (Australia).

Correspondence.

THE EXTRACTION OF CATARACT IN ITS CAPSULE (TOTAL EXTRACTION).

SIR: Dr. Kevin O'Day in his letter (THE MEDICAL JOURNAL OF AUSTRALIA, February 12, 1944) appears to be somewhat perturbed over what he terms the "display of interest in the controversy amongst those whose practice is not confined to ophthalmology", and in order to "help to clarify the issue", he proceeds to set down for their benefit and edification eight categorical "statements", the majority of which are either misleading or erroneous.

1. He would not have the method of total (intracapsular) operation described as "new", when it was only about twenty years ago that it began to gain universal recognition. When compared to the 200 year old "classical" (extracapsular) operation, it cannot be otherwise than so regarded.

2. He confuses the issue when he says that "nowhere is it more widely employed than in the Empire, particularly in India" *et cetera*. This Indian method he refers to was practised by Lieutenant-Colonel Henry Smith in the Punjab, who published his results in 1905. It is also known as "Smith's expression method" because after section the cataract was "popped" out of the eye, capsule and all, by applying pressure to the globe with the thumb or by means of a hook! The method is generally regarded as crude and unsatisfactory, so much so that "outside of India it has been adopted by comparatively few operators".[1] It is surprising therefore that Dr. O'Day should not be aware of the difference between this method and the modern one of total (intracapsular) extraction as popularized by Barraquer and Arruga of Spain, Kalt, Knapp, Torok, Elschnig and others.

3. He maintains that the "so-called immature senile cataract can be extracted by the older extracapsular method without any extra risk of failure". He is careful, however, to omit to say that this is only possible by previously submitting the patient to a "preliminary" operation in order to provoke the necessary maturation, without which the cataract cannot be extracted satisfactorily. It is only a few weeks after that the cataract would be ready for extraction.

4. "Evidence of past inflammation of the iris and ciliary body is not a contraindication to operation by this method", according to Dr. O'Day. Possibly not. But it is a definite contraindication to a successful operation, however.

5. Serious complications may not be the rule after the old operation, but a certain degree of iritis, however slight, is almost inevitable. Furthermore, posterior synechiæ, proliferation and thickening of the capsular remnants necessitating "needlings" cannot be regarded otherwise than as complications. With the new method, all these minor post-operative complications, after-treatments, needlings *et cetera* are done away with. The eye regains its normal appearance within a very short time, the pupil is clear and black, and if a complete iridectomy has not been performed, it reassumes the normal circular shape, reacts briskly to light in the normal manner, and is capable of full dilatation under a mydriatic, thus showing the absence of adhesions.

6. Although it may not be necessary to "needle every case", still a great number require this subsequent operation. Davenport,[2] analysing a series of 2,368 extractions performed at the Royal London Ophthalmic Hospital, Moorfields, found that 62·4% required needling afterwards.

Dr. O'Day, in referring to me, says that I "apparently regard an acuity of $^6/_{12}$ or better as satisfactory. This would

appear to mean that he is satisfied with a lower visual acuity". In reply to the first part of this statement I would say that Davenport (*loco citato*, page 87), for instance, regards a vision of $^{6}/_{12}$ partly or better as "good" ($^{6}/_{4}$ partly or better as "very good"). With regard to his illogical and detractory remark to the effect that I am satisfied with a lower visual acuity, one should wonder what my reason might be for acquiring and adopting a method of extraction, the technique of which the doctor himself admits "is more difficult to acquire"! He should be pleased to know, however, that there are occasions when even a very poor visual result may be satisfying to the surgeon and most gratifying to the patient.

Dr. O'Day asks: "Is it scientific or academic to draw any conclusion from the result obtained in a series of forty cases, when a balance of six is sufficient to swing the percentage of good results from 55 to 70?" I would say, in reply, that I have given no reason for anyone to think that my opinion on the advantage of the new method over the old is based on the result of this first forty cases operated on by me in Sydney. I only published them as a matter of interest in conjunction with my original article (THE MEDICAL JOURNAL OF AUSTRALIA, October 16, 1943) and for what they were worth. Actually my conclusion on the superiority of the new operation is founded on the teaching of various authorities in different parts of Europe where I had the occasion to study and to work, and also on the personal experience of some 3,000 cases operated on by me while in China. In my original article I carefully avoided to convert my results into percentages, as I was aware that from a point of view of statistics, a series of forty cases would be valueless. It was he himself who first converted them into such, and although he fully realizes that a "balance of six is sufficient to swing the percentage", yet he did not hesitate to use this very balance against my results by stating in his first letter (THE MEDICAL JOURNAL OF AUSTRALIA, November 13, 1943) that 45% of my cases regained a visual acuity of $^{6}/_{12}$ or less when, as is customary to reckon, actually 70% regained a visual acuity of $^{6}/_{12}$ and better.

Yours, etc.,

R. A. DE CASTRO BASTO, M.D., B.S., M.R.C.S., D.O.M.S.

B.M.A. House,
135, Macquarie Street,
Sydney.

February 25, 1944.

References.

(1) H. E. Fuchs: "Textbook of Ophthalmology", Eighth Edition, page 926.
(2) R. C. Davenport: "After Results of Cataract Extraction", *The British Journal of Ophthalmology*, Volume 12, 1928, page 85.

Naval, Military and Air Force.

CASUALTIES.

ACCORDING to the casualty list received on February 25, 1944, Major J. R. Cornish, A.A.M.C., Grassmere, South Australia, has been placed on the "seriously ill" list.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Wheeler, Llewellyn Daniel, M.B., B.S., 1943 (Univ. Sydney), Royal Prince Alfred Hospital, Camperdown.

Seamonds, Henry, M.B., B.S., 1943 (Univ. Sydney), Box 21, Q.V. Buildings Post Office, Sydney.

Whyte, George, M.B., B.S., 1943 (Univ. Sydney), District Hospital, Marrickville.

Yuille, Alexander Loddon, M.B., B.S., 1942 (Univ. Sydney), NX200880, Captain A. L. Yuille, H.Q., R.A.E., 6th Australian Division, A.I.F.

Lackey, Samuel, M.B., B.S., 1940 (Univ. Sydney), NX116628, 2/5th Australian Armoured Regiment, Australia.

Obituary.

VINCENT MICHAEL O'GRADY.

WE regret to announce the death of Dr. Vincent Michael O'Grady, which occurred on February 26, 1944, at Melbourne.

Books Received.

"Essentials for Final Examinations in Medicine", by John de Swiet, M.D. (London), M.R.C.P., Captain, R.A.M.C.; Second Edition; 1943. London: J. and A. Churchill Limited. 7" × 4", pp. 176. Price: 7s. 6d.

Diary for the Month.

MAR. 7.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAR. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAR. 10.—Queensland Branch, B.M.A.: Council Meeting.
MAR. 10.—Victorian Branch, B.M.A.: Ethics Subcommittee.
MAR. 13.—Victorian Branch, B.M.A.: Hospital Subcommittee.
MAR. 13.—Victorian Branch, B.M.A.: Finance Subcommittee.
MAR. 14.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAR. 14.—New South Wales Branch, B.M.A.: Ethics Committee.
MAR. 14.—Tasmanian Branch, B.M.A.: Branch Meeting.
MAR. 14.—Victorian Branch, B.M.A.: Organization Subcommittee.
MAR. 15.—Western Australian Branch, B.M.A.: Branch Meeting.
MAR. 16.—Victorian Branch, B.M.A.: Executive Meeting.
MAR. 22.—Victorian Branch, B.M.A.: Council Meeting.
MAR. 24.—Queensland Branch, B.M.A.: Council Meeting.
MAR. 28.—New South Wales Branch, B.M.A.: Council Quarterly.
MAR. 30.—New South Wales Branch, B.M.A.: Annual Meeting.
MAR. 31.—Queensland Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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